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ON BRONCHOSTENOSIS A ROENTGENOLOGICAL STUDY

by

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(Continued from last number).

3. Tuberculous changes in the bronchial walls.

Bronchostenosis occurs quite frequently in tuberculosis, in the acute as well as the chronic forms. In an earlier work based on comparative patho-anatomical and radiological investigations I gave an account of the incidence as well as pathogenesis of obstructive atelectasis. In regard to these questions the reader is therefore referred to this paper. For the present purpose I shall only give a brief review of the main results obtained.

A more or less widespread obstructive atelectasis is a very common phenomenon in acute forms of tuberculosis. Most infiltrations, that can be distinguished in acute and exudative forms of tuberculosis, are in my opinion obstructive atelectasis. In the last edition of ASSMANN's great handbook this author has published illustrations of two cases of so-called »Frühinfiltrat», which are typical cases of obstructive atelectasis. In my paper referred to above there is a great number of such cases with quite different localisations.

My anatomical investigations revealed that in these acute exudative forms of tuberculosis the bronchial occlusion was caused by a purulent exudate infiltrating and filling up the bronchial lumen on the proximal side of the atelectasis.

In my subsequent investigations into these questions I found, however, in a large number of cases the obstructive atelectasis preceded by local emphysema within the same part of the lung as the atelectasis. In these cases, therefore, the purulent exudate in the bronchus, before

completely occluding it, had only caused incomplete constriction with the result of valvular action.

Below are some cases in point.

Case 12. E. N. Fig. 21, 21 a. The patient had earlier had a left-sided pleurisy. On roentgen examination 12 months later one found a local emphysema within the posterior part of the right upper lobe, corresponding to the dorsal branch of the bronchus eparterialis. Otherwise there were no changes to be seen. Three months later this region was the seat of an obstructive atelectasis. A cavity formed within and tb. bacilli were found.

At the first examination one thus found a bronchostenosis of valvular effect of the dorsal branch of bronchus eparterialis. This stenosis probably arose through a purulent effusion which caused incomplete occlusion of the bronchus. Later a typical obstructive atelectasis arose in the same region pointing to the occlusion being completed and, finally, a cavity also formed. The cavity was then of large size and presented a rounded shape, which indicated its being under valvular action.

Case 13. L. O. Fig. 22. Earlier in good health. Taken ill one week ago with rise of temperature of up to 39°C. There was dulness over right apex and a few harsh râles in I. 2 on the right side. S. R. 56 mm. On roentgen examination there were found in the posterior part of the right upper lobe a few well localised dense patches. In the anterior portion of the upper lobe, corresponding to the anterior part of bronchus eparterialis, there was seen a wedge-shaped clearer area with convex outline besides a narrow wedge-shaped massive shadow. The latter was located immediately below the former. Later on obstructive atelectasis also arose in this region.

The rtg. examination has thus shown not only signs of healed tuberculous foci in the posterior part of the right upper lobe but also signs of bronchostenosis of the anterior branch of bronchus eparterialis. The lower branch of this bronchus is completely occluded with obstructive atelectasis of corresponding lung area. The upper branch is partly constricted with valvular action; corresponding lung area being the seat of secondary emphysema and anaemia. These constrictions would seem caused by recent tb. changes.

Case 14. S. B. Fig. 23. On roentgen examination of this case one found two seats of obstructive atelectasis corresponding to the distributive areas of the ventral and dorsal branches of bronchus eparterialis besides a local emphysema corresponding to the apical branch of the same bronchus. Later on obstructive atelectasis also arose in this region.

In this case there was thus complete occlusion of the ventral and dorsal branches of bronchus eparterialis and incomplete stenosis of the apical branch.

Comments. These cases show that tuberculosis in the acute stage may begin with but incomplete occlusion of a bronchus with secondary emphysema and anaemia within a local area of the lung. It is only later

Fig. 21. Case 12. There is obvious emphysema and anaemia in the upper and posterior parts of the right upper lobe. Signs of old left-sided pleurisy.

Fig. 21 a. Same case 2 months later. Massive opacity of upper and posterior parts of upper lobe. Within this can be seen a lighter area, at least the size of a walnut, with convex outline.

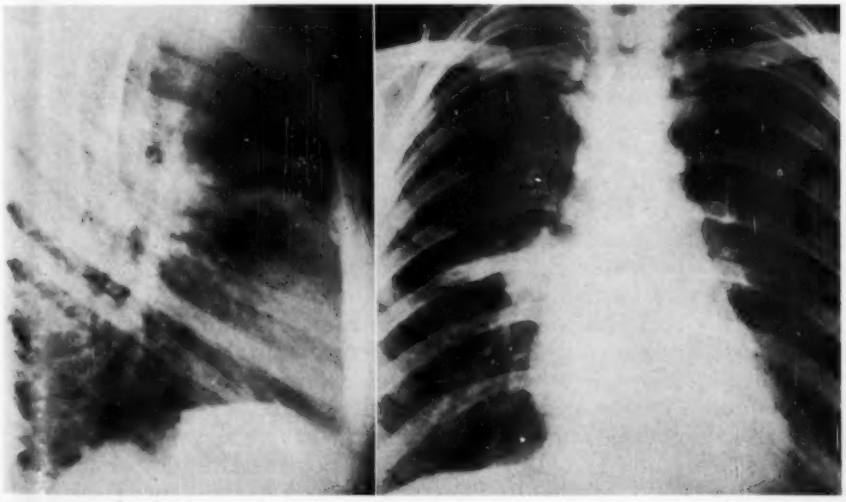

Fig. 22. Case 13. In the posterior part of the right upper lobe there is a number of well defined dense patches. In the anterior part of the same lobe, corresponding to the anterior branch of bronchus eparterialis, there is a wedge-shaped region with convex outline and well marked emphysema and anaemia and below this another wedge-shaped area massively infiltrated and with a concave outline.

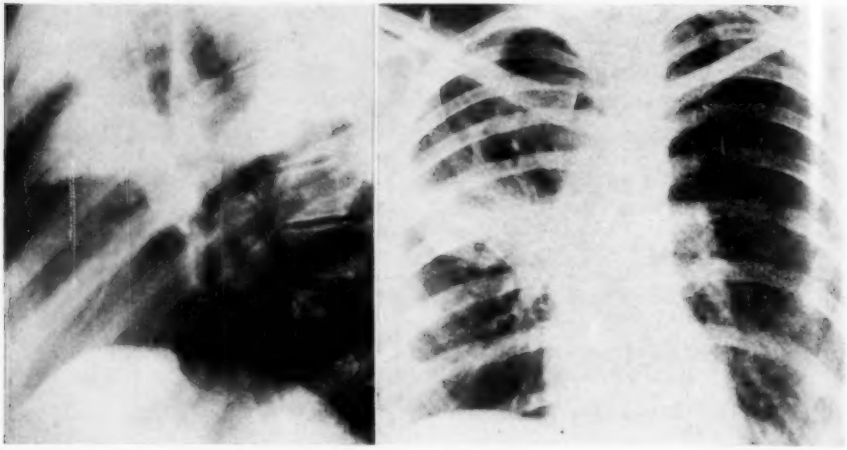

Fig. 23. Case 14. In the upper lobe we discern two wedge-shaped massive opacities with concave bounding surfaces, corresponding to the distributive areas of the ventral and dorsal branches of bronchus eparterialis. In the upper part of the upper lobe, corresponding to the apical branch, there is obvious emphysema and anaemia.

that complete occlusion with obstructive atelectasis arises in these cases. In some cases, therefore, a local emphysema may be the first roentgenological sign of early tuberculosis.

In these acute cases the bronchostenosis is usually localised to one or two adjacent lateral bronchi. The bronchostenosis with its consequences, emphysema and atelectasis, runs a course in these cases which is entirely dependent upon the progress of the original disease. In some cases it may thus entirely disappear if the original disease has cleared up or else it may leave slight scars behind. In other cases the tuberculous process may continue in the atelectatic area. This renders the atelectasis not a pure one; the alveoli get expanded by the exudate whereby the reduction in volume becomes less marked. In these cases, however, further atelectases usually arise in the surroundings through new bronchi becoming occluded by the tuberculous purulent exudate.

Sooner or later the tuberculous purulent exudate undergoes caseous necrosis and a cavity forms. If this cavity formation is located on the peripheral side of the bronchial occlusion the cavity as well as surrounding lung tissue are devoid of air and will not therefore appear on the radiogram. It is only when the caseous necrosis has advanced as far as an air-carrying bronchus that the cavity will contain air and thus appear on the radiogram. In these early stages the bronchus leading to the cavity is usually more or less constricted wherefore valvular action o-

tai s. Air is then forced into the cavity which becomes expanded and ass mes a rounded shape. Should the valvular action be pronounced the cavity may rapidly enlarge from the increased pressure within. Should the bronchostenosis subsequently become less marked the cavity will als be reduced in size. A rounded cavity increasing in size does not the efore generally mean progressive liquefaction. In the case of the ste osis being completely restored the cavity will assume the form and size accorded it by the liquefied parenchyma. As the liquefaction does not always take place concentrically a cavity whose afferent bronchus is not constricted assumes in irregular shape. A rounded cavity is thus an indication of bronchostenosis. Should the tuberculous process subsequently undergo progression the afferent bronchus of the cavity may again become occluded. The air then disappears from the cavity which is thus no longer to be seen in the radiogram. Disappearance of a cavity on the radiogram does not mean, therefore, in these cases that the process has healed but rather indicates aggravation of the condition.

In chronic cases of pulmonary tuberculosis a multiple condition of bronchostenosis occurs very frequently and is generally very widespread. Such multiple affections are caused by changes brought about by the tuberculous bronchitis. In judging the roentgen appearance of a chronic pulmonary tuberculosis it is necessary therefore to bear in mind the changes to which bronchostenosis may lead. All the three stages of bronchostenosis may occur in chronic pulmonary tuberculosis. Since in this disease it is generally a question of stenosis of smaller bronchial branches, the individual changes will mainly come to be located in the periphery of the lung and occupy less space within the lung. They appear as small opacities, wedge-shaped by appropriate projections, or as wedge-shaped or vesicular attenuations. The changes are subject to very great variation and may vary from one examination to another. By the fact that often a number of adjacent bronchial branches are affected, the individual changes however may become confluent and thus larger parts of the lung become altered. Frequently, however, there are different degrees of stenosis in the individual bronchial branches whereby atelectatic opacities and emphysematous attenuations will come to alternate with one another. Atelectatic opacities passing into emphysematous attenuations within the same lung area at different examinations generally means progressive necrosis of the changes in the tuberculous bronchus, while a change the other way round from one occasion to another means acutisation of the process with increasing exudative changes in the bronchi.

I would lay particular stress upon the emphysematous 'clearing-up' in judging the roentgen picture in chronic pulmonary tuberculosis

since so far too little attention has been paid to them and they probably are to be blamed for most misinterpretations.

In some cases an emphysematous clearing-up on the radiogram may almost completely neutralize the changes produced by the tb. process, not only within the emphysematous area but also within regions located in front or behind the emphysematous clearing-up on an antero-posterior picture. Such changes will however appear on a lateral picture.

As instance of this condition the following case may be quoted.

Case 15. E. L. Fig. 24. This patient had for years suffered from pulmonary tuberculosis. Has latterly grown worse. On roentgen examination one then found in an antero-posterior view a clearly bullous emphysema in the right upper lobe. The emphysema is bounded below by a convex band-shaped opacity. Otherwise there were no shadows to be seen except close to the hilum where some streak-shaped shadows could be observed. On lateral pictures, on the other hand, the posterior and upper part of the upper lobe are seen to be occupied by large vesicular attenuations, while the anterior part of the upper lobe shows well marked opacities with an extensive cavernous system made up of numerous cavitations, the size of hazel-nuts to beans, surrounded by opaque marginal zones. On tomographic examination the emphysema appeared as a large rounded attenuated area that might be mistaken for a cavity. On clinical examination the patient presented obvious signs of cavitation in I. 1 in front while the breath-sounds were much impaired behind. Nor were any râles to be heard at the back.

This case then shows signs of a well marked bullous emphysema of the dorsal and apical parts of the upper lobe besides a cavernous process of the anterior portion of the upper lobe. The bullous emphysema has almost completely neutralize the changes in the anterior part of the upper lobe, rendering these invisible on antero-posterior films.

Another very common source of error is, as was first pointed out by LAURELL, that larger or smaller emphysematous vesicles, surrounded by an opaque marginal zone, are mistaken for cavitations.

4. Changes in the bronchial wall as a result of cancer.

Bronchial cancer itself does not generally appear on the radiogram until it has reached a very advanced stage. Since however it produces, already at an early stage, a more or less marked bronchostenosis, it is possible thereby to be able to make an early diagnosis by roentgen examination. I have earlier described 46 cases of cancer bronchiale, in a paper under the title »Bronchial Carcinoma, a Roentgen-Pathological Study;» since then I have seen another 7 cases.

Case 16. J. B. Fig. 25, 25 a, 25 b, 25 c. The first examination of this case revealed a local emphysema corresponding to the first dorsal branch of bronchus hyparterialis. The film thus shows a definite clearing-up within the part of the lung that corresponds to this bronchus; it presents a convex outline and is bounded by a thin denser marginal zone. On examination 2 months later there was found within the same area a wedg

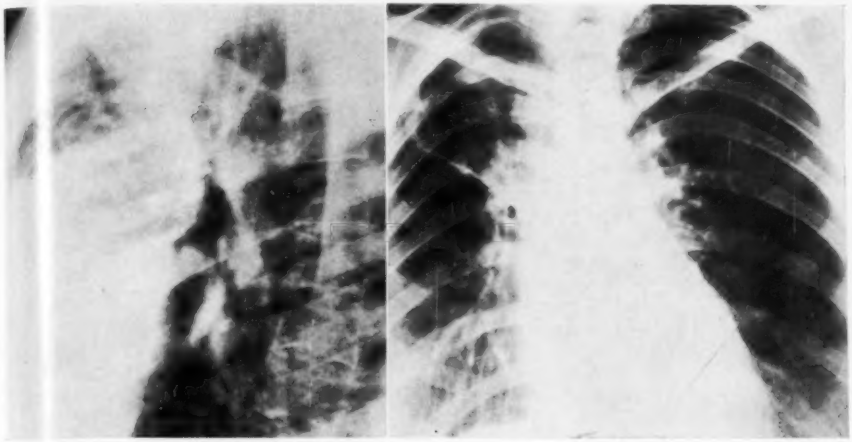

Fig. 24. Case 15. There is obvious emphysema with a convex border in the posterior part of the upper lobe and in the anterior part of this there is a definitely dense region within which it is possible to distinguish a number of attenuated areas, the size of about hazelnuts, (cavities). On an antero-posterior picture the infiltrated region is seen to be almost obliterated by the emphysematous area.

shaped massive shadow, the area occupying considerably less volume than earlier. The shadow also corresponds to the same bronchial branch. Bronchography showed the first dorsal branch of bronchus hyparterialis to be constricted immediately after its origin. The stenosis shows a torn and jagged outline. The diagnosis was verified by bronchoscopy and exploratory excision. The patient was subsequently operated on and is still living free from symptoms 3 years after the operation.

Case 17. M. L. Fig. 26. This case shows a local emphysema in the anterior lower portion of the left upper lobe. It remained unchanged for a couple of months. Bronchography was carried out which showed local changes within the anterior lower branch of the bronchus of the left upper lobe, which presented a torn and jagged outline over an area of about 1 cm. The result seems to have been slight stenosis of the bronchial lumen. The diagnosis was verified by bronchoscopy.

Case 18. L. L. Fig. 27. This case shows local emphysema, particularly in evidence during expiration, of the upper posterior portion of the right lower lobe. On bronchography the first dorsal branch of bronchus hyparterialis appeared constricted in a cornet-like fashion. On bronchoscopy a bleeding tumour was found in this bronchus.

Case 19. E. S. Fig. 28. This case presents a definite emphysema with convex outline within the anterior upper part of the left upper lobe. In the hilum there is a large mass of glands with irregular nodular outlines. Spontaneous pneumothorax subsequently arose. The upper lobe was then seen to be atelectatic while the lower lobe contained a good amount of air. The patient subsequently died and proved to have an infiltrating tumour within the left upper lobe.

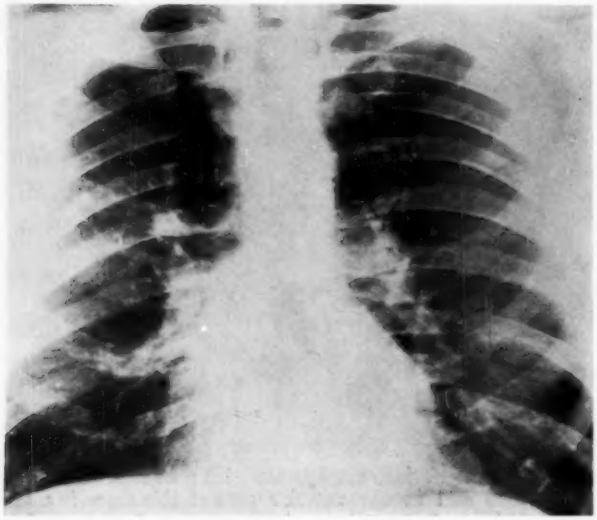

Fig. 25. Case 16. In the posterior upper part of the lower lobe, corresponding to the first dorsal branch of bronchus hyparterialis dx., there is obvious emphysema with anaemia; its limiting border is convex.

Case 20. K. K. Fig. 29. This case shows a wedge-shaped massive opacity of the posterior lower part of the right lower lobe. On the other hand the anterior lower part of the middle and lower lobes show signs of well marked emphysema with convex outlines. There is thus complete occlusion of the dorsal branches of bronchus hyparterialis dx. and a valvular stenosis of the ventral branches. This was also verified by bronchography, which showed a cornet-shaped torn defect of bronchus hyparterialis with complete occlusion of the posterior branches. The anterior branches, on the other hand, leading to the anterior lower part of the middle and lower lobes, were seen to be filled. At their origin, however, these branches present a circular stenosis which continues for a few millimetres into the bronchi.

Comments. Out of the 53 cases of bronchial cancer that I have seen all but one have shown signs of bronchostenosis as the first symptom. The case that did not show any bronchostenosis had a well localised tumour of the right lower lobe, in the periphery of the lung.

In more than 25 % or in 15 cases there were signs at the first examination of valvular stenosis with local emphysema and anaemia of the lung area corresponding to the affected bronchus. In the majority of these cases there subsequently arose obstructive atelectasis of the same lung area.

The other 37 cases presented already on the first examination obstructive atelectasis.

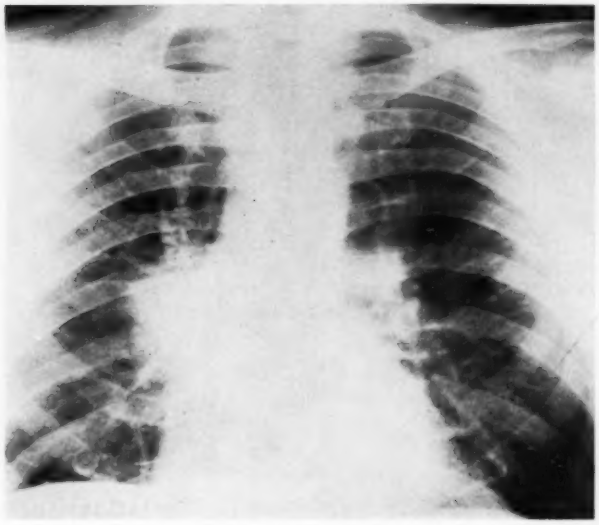

Fig. 25 a.

Fig. 25 b.

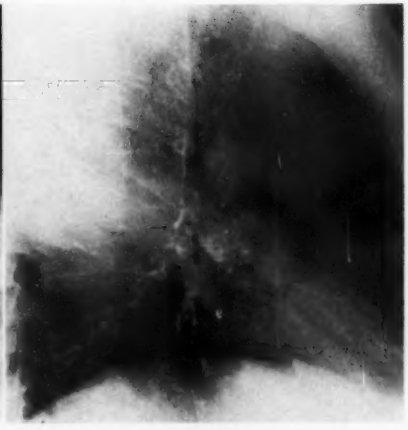

Fig. 25 c.

Fig. 25 a and b. Same case 2 months later. In the upper posterior part of the lower lobe, corresponding to the first dorsal branch of bronchus hyparterialis, there is now a massive wedge-shaped opacity with concave bordering surfaces.

Fig. 25 c. Same case after bronchography. The first dorsal branch of bronchus hyparterialis dx. is seen to be occluded. The occlusion presents an irregular and torn outline.

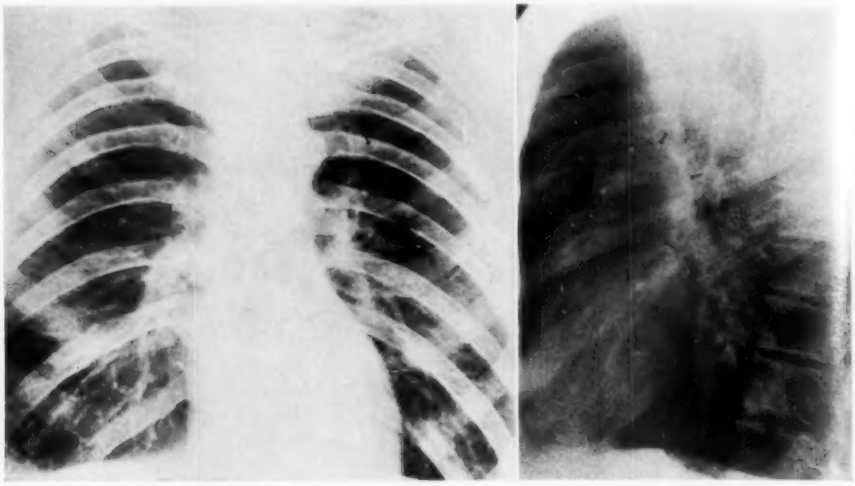

Fig. 26 and 26 a. Case 17. The left upper lobe, corresponding to the upper anterior branch of the bronchus, is the seat of distinct emphysema with anaemia. The emphysematous area has a convex border.

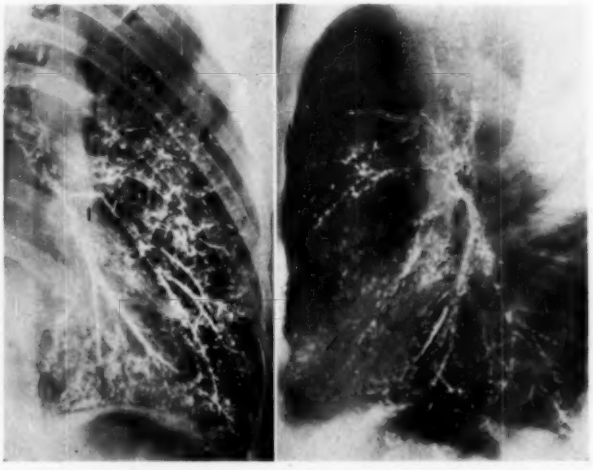

Fig. 26 b and c. Same case after bronchography. About 1 centimetre from its origin from the left bronchus hyparterialis the first ventral branch is seen to have a torn and jagged outline. The defect extends forwards towards the anterior upper branch which is seen somewhat constricted.

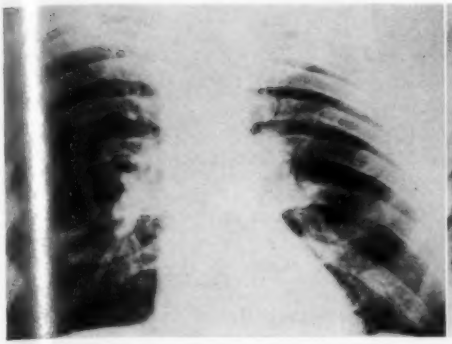

Fig. 27. Case 18. The posterior part of the right lower lobe, corresponding to the second dorsal branch of bronchus hyparterialis, is the seat of obvious emphysema with anaemia. The emphysema has a definitely convex outline.

Fig. 27 a. Same case during expiration. Emphysema appears much more distinct.

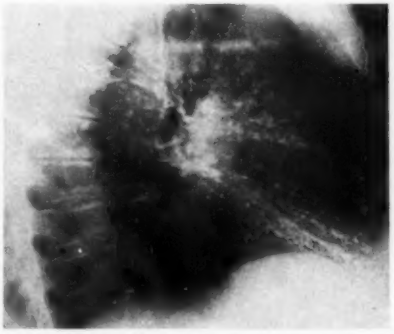

Fig. 27 b. Same case after bronchography. The second dorsal branch of bronchus hyparterialis dx. is seen to be constricted in a cornet-shaped manner.

At the bronchographic examination the infiltration proved in all the cases to be coherent. In earlier cases the infiltration was located in a bronchial branch of the first to the third degree. In but 2 cases was it primarily localised to a main bronchus. By careful observation of the cases for a prolonged period of time, in several cases for more than 2—3 years, I have been able to observe that the cancer does not only grow into the lung in a peripheral direction, as formerly assumed, but also in a central direction into and around the bronchial wall. During this central growth the cancer will also infiltrate more centrally situated adjoining bronchi. In these bronchi it has also in some cases extended in a peripheral direction. A picture can then be obtained as in case 20, fig. 29, with an obstructive atelectasis in one part of the lung and a local emphysema in another. Thus signs of different stages of bronchostenosis in different bronchi. This pseudo-multiple localisation of cancer differs, however, from multiple stenosis present in inflammatory changes in the bronchi by the fact that in the former case it is always

Fig. 28 and 28 a. Case 19. The left upper lobe, corresponding to the anterior upper branch of the first ventral branch of bronchus hyparterialis sin., is the seat of obvious emphysema with anaemia. The emphysematous area has a convex outline and in the left hilum can be seen a large nodular glandular shadow.

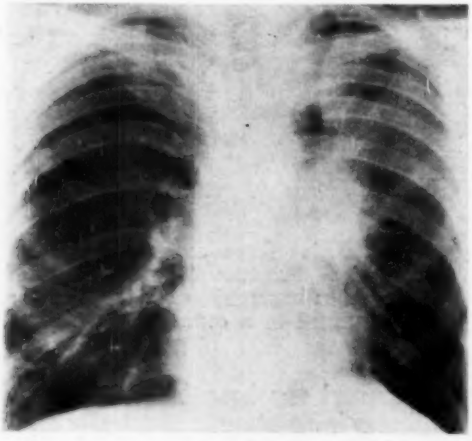

Fig. 28 b. Same case 4 months later. Spontaneous pneumothorax has formed. The upper lobe is massively opaque (atelectasis). The lower lobe shows good aeration.

adjacent bronchi that are subject to changes. In bronchography of cancer bronchiale we also always find confluent infiltration of the bronchi with stiff and torn outlines while in inflammatory stenosis there are multiple and separate constrictions with smooth and rounded contours.

Bronchial cancer seems later to have become relatively more common in Sweden than formerly. In my own material gastric cancer is thus $6^1/_2$ times as common as bronchial cancer while in NYSTRÖM's material from 1911—1912 gastric cancer occurred 74.5 times as often as cancer of the lung. Other statistics too show similar figures. According to a great statistical account from Basel based on autopsic material cancer of the stomach

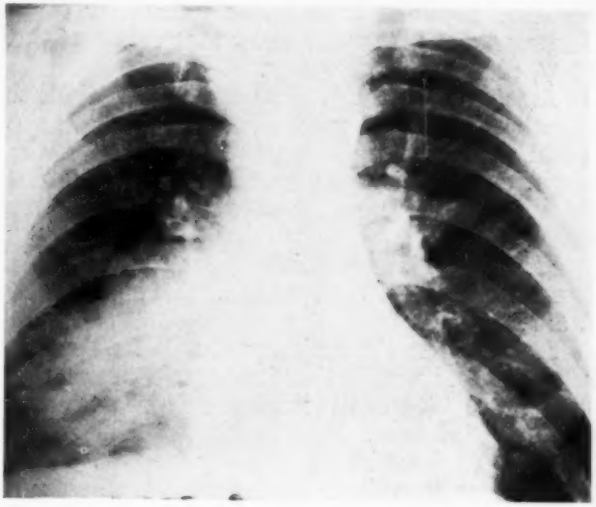

Fig. 29 and 29 a. Case 20. In the posterior lower part of the right lower lobe there is a massive wedge-shaped opacity with concave outlines. In the anterior lower part of the same lobe and in the middle lobe there is obvious emphysema with anaemia. This area presents a clearly convex outline.

Fig. 29.

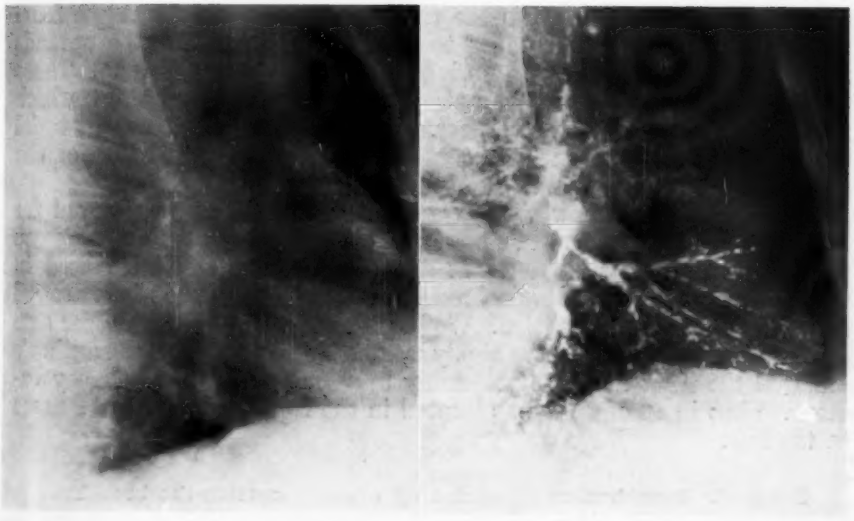

Fig. 29 a. Fig. 29 b.

Fig. 29 b. Same case after bronchography. Bronchus hyparterialis is the seat of a distinct defect with torn outline. The dorsal branches below the first dorsal bronchial branch are completely occluded. The two anterior upper branches show a circular stenosis with an irregular lumen which permits the oil to pass through.

occurred 16 times as often as pulmonary cancer. New statistics by FERENZY and MAROLCZY show that cancer of the lung makes up about 10 % of all forms of cancer.

Among the author's cases no less than half the number of patients have been under 50 years of age. The mean age for bronchial cancer would thus seem to be somewhat lower than the mean age for cancer in general. This is also pointed out by LENK. Earlier statistics however show the age for bronchial cancer to be the same as for cancer generally.

Most statistics are pointing out that bronchial cancer occurs much more often in men than women. The ratio men to women in my cases is 5: 1.

According to the majority of authors the right lung is supposed to be the seat of predilection for bronchial cancer (KAUFFMANN and LENK and others) and of the lobes the upper ones are said most often to be the seat of disease. Most authors also state that a main bronchus is generally the seat of the primary tumour. It is thus stated by WOLF and KIKUTH that the main bronchus is the seat of the primary tumour in no less than one-third of the cases. Cases not having originated from the main bronchi are considered having done so from one of the larger side bronchi. More seldom is the cancer supposed to take origin from the smaller bronchi. According to these authors the growth spreads from the point of origin in a peripheral direction into the lung. These observations have been made, however, on an autopsic material, which shows but the final stages of cancer. It is impossible from such material to draw any definite conclusions as to the primary localisation of the growth or its distribution in the lung.

My own material however is largely made up of cases observed for a longer or shorter period of time during life, from the time the disease was first diagnosed till death. It shows the cancer to be localised in most cases to one of the finer bronchi. From here it grows in a central as well as peripheral direction. While growing centrally the cancer also extends secondarily into more highly located side bronchi. In the author's material the right lung has preferably been the seat of the primary tumour. Thus in 40 cases it was located in the right lung and in 13 cases in the left.

Group C. Bronchostenosis caused by changes outside the bronchus by which this is compressed

1. *Enlarged lymph-nodes.*

Enlarged glands in the hilum often give rise to a more or less pronounced bronchostenosis. In early stages there is generally stenosis o

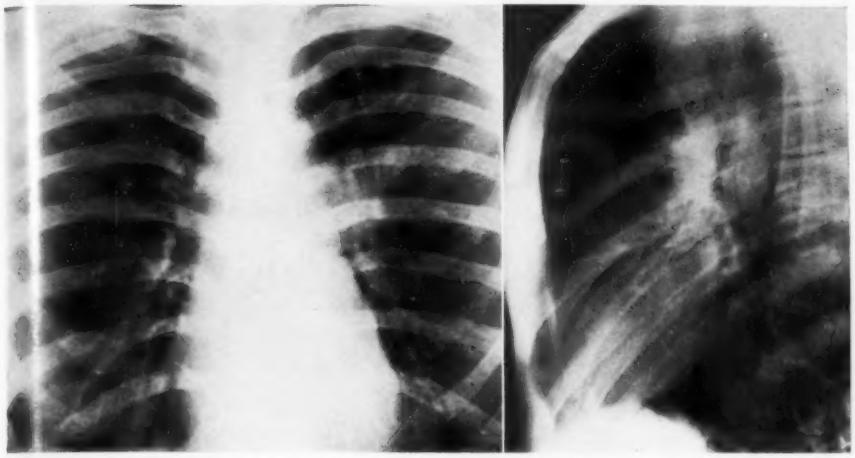

Fig. 30. Case 21. A large glandular mass with ill-defined outlines in the left hilum. The anterior portion of the left upper lobe, located immediately in front of the large glandular shadow, is the seat of definite emphysema with anaemia. The emphysematous area shows a convex outline.

a milder degree but in more severe cases complete occlusion may arise, most often leading to lobar atelectasis, to a greater or lesser extent.

Case 21. M. C. Fig. 30. This case presents a gland, the size of a walnut, in the left hilum, located above the first ventral branch (bronchus of upper lobe). In the anterior part of the upper lobe there is distinct emphysema with anaemia. This emphysematous area shows a convex outline and is bounded above by a thin band-shaped denser zoon.

This is a case then of hilar adenitis with valvular stenosis of the anterior branches of the bronchus of the upper lobe, clearly caused by compression of these bronchial branches.

Case 22. T. S. Fig. 31. This case shows a shadow of a gland, the size of about a walnut, in the right hilum, in the anterior and lower part of this. The midlobe seems emphysematous and is definitely expanded with a convex outline. The vascular design is less marked than normal in this lung. On examination 2 months later the hilar gland appears as before. Now however there is obstructive atelectasis of the lateral lower part of the middle lobe, while emphysema remains in the middle lower part of this lobe.

Bronchography was carried out 20 days later. The atelectasis and emphysema in the middle lobe persist unchanged. The bronchus of the midlobe is compressed and presents a soft depression on its upper surface; this gives the bronchus the shape of a comet. The gland seems to be lying immediately above this stenosis.

On examination 12 months later the atelectasis had completely disappeared but there was now well marked bronchiectasis of the midlobe.

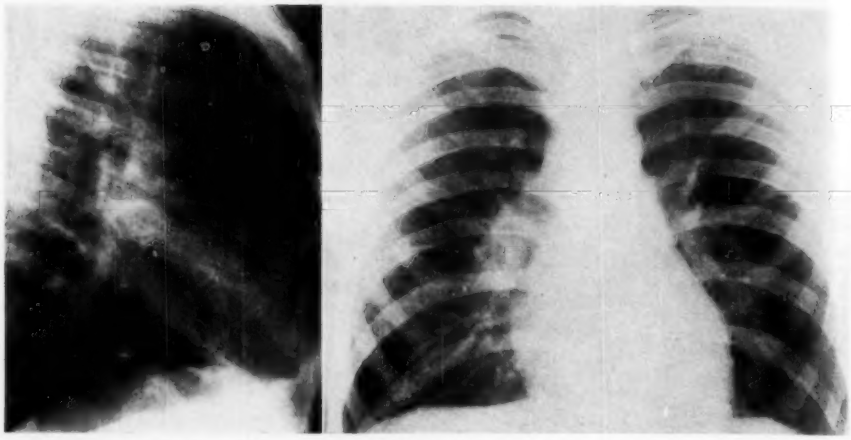

Fig. 31 and 31 a. Case 22. In the right hilum can be seen a large rather poorly defined glandular mass. The midlobe is seen to be much expanded and presents definite emphysema and anaemia.

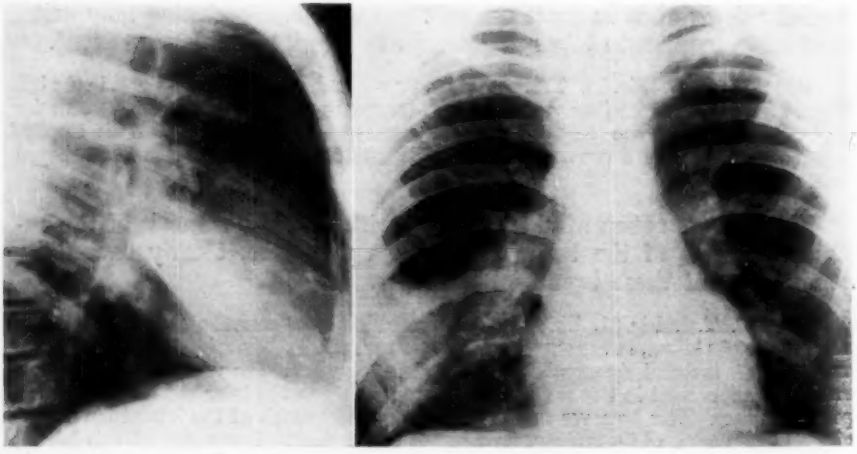

Fig. 31 b and c. Same case 2 months later. Glandular mass as before. In the lateral part of the middle lobe there is a massive wedge-shaped opacity with a downward concave bordering surface and in the medial part of the same lobe there is a definite emphysematous area.

This case thus shows a hilar adenitis which had produced a valvular stenosis increased causing obstructive atelectasis to arise in the part of the middle lobe corresponding to the lateral lower branch of the bronchus of this lobe. After 12 months during which the obstructive atelectasis remained unaltered well marked bronchiectasis arose in the middle lobe.

Fig. 31 d. Same case after bronchography. The first ventral branch of bronchus hyparterialis dx. is seen to be occluded. It has the shape of a cornet. The bronchial branch is seen close to the glandular shadow described.

Comments. Hilar adenitis often leads to bronchostenosis of various degree with secondary emphysema or atelectasis of a local area of the lung corresponding to the bronchus compressed by the gland. Since these glands are located in the centre the changes produced will generally be localised to a lobe or the greater part of a lobe.

In order to form an opinion of hilar adenitis and its prognosis it is exceedingly important to recognise these emphysemata and atelectases. The latter are often mistaken for parenchymatous infiltrations and the former are often overlooked.

If an obstructive atelectasis should take a chronic course as a result of hilar adenitis bronchiectasis will often arise (see case 22) as in other cases of chronic obstructive atelectasis. There is quite a number of such cases in the author's material. Hilar adenitis with secondary obstructive atelectasis would seem to be a very frequent cause of acquired bronchiectasis even in early childhood.

2. *Intrathoracic tumours and aneurysm.*

Case 23. G. Ö. Fig. 32. The patient is under treatment for a lymphosarcoma in the right supraclavicular fossa. On earlier roentgen examinations lung findings were normal. Patient gradually began to suffer from shortness of breath and irritable cough. On roentgen examination then undertaken one found a mass of glands located paratracheally on the right side and extending down towards the hilum. The right upper lobe was mildly atelectatic with a concave lower outline. This atelectasis was very evident during inspiration but not so much so during expiration. Two months later there was an emphysema and six months later an obstructive atelectasis in the upper lobe.

The roentgen examination has thus shown signs of a glandular mass in the right mediastinum which first gave rise to mild bronchostenosis of bronchus eparterialis with secondary mild atelectasis of the right upper lobe. Later on more advanced stages of bronchostenosis appeared.

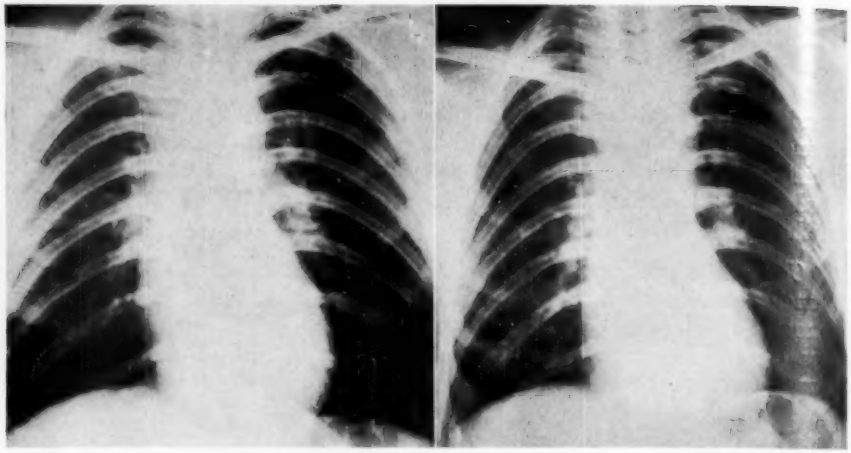

Fig. 32. Case 23. Mass of glands paratracheally on the right side. The upper lobe shows slight atelectasis. The lower bordering surface of the lobe is concave. During inspiration there is some displacement of heart towards the affected side. During expiration the atelectasis is still apparent though not so obvious. The displacement is inhibited.

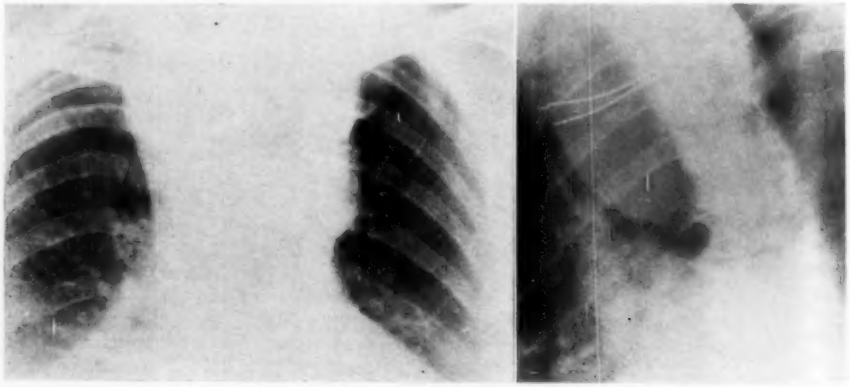

Fig. 33. Large intrathoracic tumour in the upper mediastinum. Trachea is seen displaced to the right and the left main bronchus downwards as a result of the tumour. The left main bronchus is clearly compressed. The entire left lung is the seat of definite emphysema and anaemia.

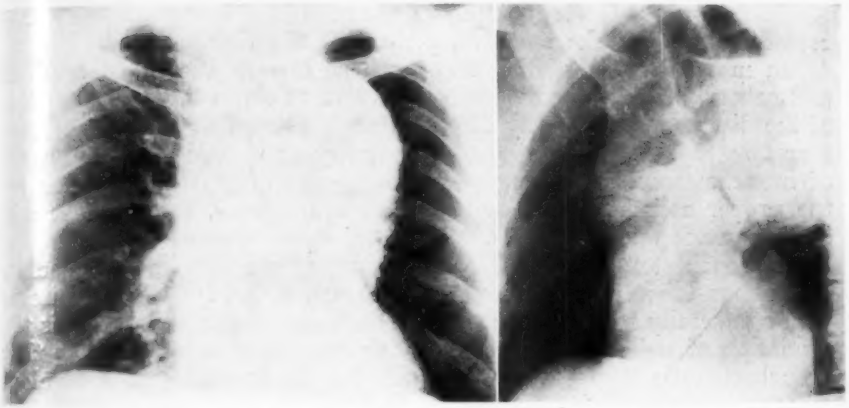

Fig. 34. Large aneurysm of descending aorta. It seems to push downwards the left main bronchus, compressing it. The left lung presents definite emphysema and anaemia.

Case 24. H. P. Fig. 33. Large intrathoracic tumour located in the left anterior mediastinum. Trachea is displaced towards the right. The left main bronchus is displaced downwards and clearly compressed. The whole of the left lung is clearly emphysematous and anaemic.

I have earlier published a case of lymphogranulomatosis with a large mass of glands located in the anterior part of the right mediastinum extending down towards the bronchus eparterialis. This bronchus was clearly occluded. The upper lobe was the seat of well marked obstructive atelectasis.

Case 25. A. L. Fig. 34. Large aneurysm of descending aorta, pushing and compressing the left main bronchus. The whole of the left lung is clearly emphysematous and anaemic.

Comments. Intrathoracic tumours and aneurysm often produce more or less pronounced bronchostenosis. This stenosis is generally localised to a main bronchus but can also be located in one of the larger and central side branches. In these cases the emphysema or atelectasis will thus come to include an entire lung or a lobe of the lung.

With this work I have wished to stress the importance of a refined roentgen diagnostics in bronchostenosis. Since bronchostenosis may occur in any kind of pulmonary condition and, moreover, is of very frequent occurrence it behaves us at every radiological examination of the lungs to take into consideration the possible changes that the stenosis may produce, if we are to obtain a correct interpretation of the roentgen appearance in the special cases.

On account of the topography of the bronchi it is essential for a correct diagnosis to take a lateral picture of the thorax.

In many diseases of the lung, such as foreign bodies, tuberculosis and cancer, bronchostenosis is the first and at times the only symptom of the disease. For an early diagnosis of these conditions it is necessary therefore to be able to recognise the varying radiological appearance of bronchostenosis. A bronchostenosis is also of great importance for the proper estimation of cavities, their appearance, development and disappearance on radiograms.

A bronchostenosis caused by inflammatory changes in the bronchial wall of non-tuberculous nature is to be regarded as a typical lung disease with a generally progressive course. The final stage of the disease is the formation of well marked bronchiectasis. It would seem therefore that bronchostenosis is of great importance for the formation and development of bronchiectasis.

In most cases, however, it is impossible in an ordinary roentgen examination to be sure of the cause of the bronchostenosis. To this end it is necessary to carry out bronchography and in my view this is an essential undertaking in all cases of chronic bronchostenosis of doubtful origin. With the technique employed nowadays at St. Görans' Hospital the risk of this performance would also seem to be very slight.

The roentgenological and bronchographic examination should subsequently be supplemented by bronchoscopy.

An early and correct diagnosis of bronchostenosis may also enable earlier and more active therapy to be adopted in a great many pulmonary diseases.

Conclusions

Bronchostenosis is a very common phenomenon in different kinds of pulmonary disease. From a radiological point of view it can be divided into 3 stages:

Stage 1 shows mild atelectasis besides hyperaemia of the lung area corresponding to the constricted bronchus.

Stage 2 shows local emphysema and anaemia of the lung area corresponding to the constricted bronchus.

Stage 3 shows massive pulmonary collapse (obstructive atelectasis) of the area corresponding to the constricted bronchus.

Between the first and third stage there is a transitory stage with inspiratory atelectasis and expiratory emphysema.

The anatomical distribution of the bronchi causes the changes in suitable projections to appear wedge-shaped.

With the changes present in stages 1 and 3 the pulmonary region corresponding to the constricted bronchus occupies a smaller volume than normal. The wedge-shaped shadows then often assume a concave outline.

Should the reduction in volume be considerable we also get displacement of the heart and mediastinum towards the affected side, retraction of the thoracic wall and elevation of the diaphragm.

With the changes present in stage 2 the lung region corresponding to the constricted bronchus occupies a larger volume than normal. The wedge-shaped cleared-up area will then obtain a convex outline. In advanced forms the changes assume a spherical shape.

In the case of a greatly increased volume we also get displacement of heart and mediastinum to the healthy side, expansion of the thorax and depression of the diaphragm.

Since the reduction in volume in stages 1 and 3 is more marked during inspiration than in expiration and the increase in volume in stage 2 is greater in expiration than inspiration respiratory oscillation of heart and mediastinum will take place in all the stages.

A bronchostenosis may arise from: changes in the lumen, changes in the bronchial wall and changes outside the lumen.

Group A. Bronchostenosis caused by changes in the lumen.

This group includes cases caused by plugs of mucus or pus, blood and foreign bodies. Such changes, emphysema and atelectasis, that arise through secretion are very transient and run an acute course.

In the presence of foreign bodies the changes may assume a chronic course unless the foreign body be found and removed.

Group B. Bronchostenosis caused by changes in the bronchial wall.

1. By inflammatory changes in bronchitis and bronchiectasis.

These forms make up a typical pulmonary disease running a chronic progressive course with acute exacerbations, accompanied by aggravation and progression of the ectatic formation. The terminal stage is characterised by well marked bronchiectasis and more or less clearly defined atelectasis.

2. By inflammatory changes in pneumonia.

Pneumonia can be divided into two types. One of them is combined with atelectasis, the so-called obstructive pneumonia. In this type the bronchi of the first or second order are occluded by fibrinous purulent exudate. It is characterised roentgenologically by a wege-shaped larger or smaller massive opacity occupying part of a lobe or in some cases an entire lobe or several lobes. The altered lung area occupies a smaller volume than normal.

The second type, the so-called true pneumonia, is not combined with any obstructive atelectasis. This form shows no occlusion at all or only such of the finest bronchial branches. It is characterised, roentgenologically, by one or several lobes being massively opaque. It is generally possible to distinguish air-carrying bronchi within an opaque lung area. The altered lung region occupies a larger volume than normal.

Obstructive pneumonia does not present the same sudden onset as true pneumonia besides running a milder course than this in uncomplicated cases.

3. By tuberculous changes in the bronchi.

Obstructive atelectasis is a very common phenomenon in acute tuberculosis. Thus most acute »infiltrations» are obstructive atelectasis.

Acute tuberculosis may also begin, however, with a local emphysema by complete bronchostenosis having failed to arise, a valvular stenosis having instead formed. Such

emphysema will generally later pass into an obstructive atelectasis by the stenosis becoming complete. The recognition of this local emphysema is of great importance for the early diagnosis of tuberculosis.

Bronchostenosis of different degrees and distribution is of very common occurrence in chronic tuberculosis. For a right judgment of the progress of the disease the changes produced by these forms of bronchostenosis are very important.

Disappearance of a cavity on the radiogram does not necessarily mean its subsidence but instead generally indicates aggravation of the condition, in that the bronchus leading to the cavity has become occluded by a fresh purulent exudate.

A rounded cavity varying in size is indicative of stenosis with valvular effect of the bronchus leading to the cavity.

Local emphysema may be mistaken for a cavity.

4. By cancerous infiltration of the bronchi.

Bronchial cancer does not generally appear on the radiogram until at a very late stage. Since however bronchial cancer already at an early stage produces bronchostenosis we are able also at this stage to diagnose the condition. For the diagnosis in question it is necessary to carry out bronchography. This examination must be carried out in all cases of chronic atelectasis in order to exclude or confirm the presence of cancer. The growth appears here as a confluent defect with torn and stiff outlines.

Group C. Bronchostenosis caused by changes outside the bronchus by which this becomes compressed wholly or in part.

1. Enlarged lymphomata.

Enlarged lymph nodes generally appear straight away on the radiogram. Thanks to their position the first side branches generally become compressed whereby secondary lung changes, emphysema and atelectasis, come to occupy the major part of a lobe or an entire lobe.

2. Mediastinal tumours and aneurysm.

These also as a rule appear straight away on the radiogram. Thanks to its position a main bronchus or in some cases the first and more centrally located side bronchi will generally become compressed whereby the secondary lung changes, emphysema and atelectasis, come to occupy an entire lung or lobe.

SUMMARY

Bronchostenosis is a very common phenomenon in different kinds of pulmonary disease. From radiological point of view it can be divided into 3 stages: Stage 1 shows mild atelectasis and hyperaemia, stage 2 shows local emphysema and anaemia and stage 3 shows massive pulmonary collapse (obstructive atelectasis). The alterations are located to the lung area corresponding to the constricted bronchus. Between the first and second stage there is a transitory stage with inspiratory atelectasis and expiratory emphysema.

The anatomical distribution of the bronchi causes the changes in suitable projections to appear wedge-shaped. With the changes present in stages 1 and 3 the pulmonary region corresponding to the constricted bronchus occupies a smaller volume than normal. The wedge-shaped shadows then often assume a concave outline. With the changes present in stage 2 the lung region corresponding to the constricted bronchus occupies a larger volume than normal. The wedge-shaped cleared-up areas then obtain a convex outline. In advanced forms the changes assume a spherical shape. Since the reduction in volume

in stages 1 and 3 is more marked during inspiration than in expiration and the increase in volume in stage 2 is greater in expiration than inspiration respiratory oscillation of heart and mediastinum will take place in all the stages.

A bronchostenosis may arise from changes in the lumen, changes in the bronchial wall and changes outside the lumen.

The author gives an account of the occurrence and course of bronchostenosis in different kinds of lungdisease and discusses the importance for a correct interpretation of the radiogram and the complications that may be produced.

ZUSAMMENFASSUNG

Bronchostenose ist ein sehr häufiges Phänomen bei verschiedenen Arten von Lungenleiden. Vom radiologischen Standpunkte lassen sich 3 Stadien unterscheiden: Stadium 1 zeigt leichte Atelectase nebst Hyperämie, Stadium 2 zeigt lokales Emphysem und Anämie und Stadium 3 zeigt massiven Lungenkollaps (obstruktive Atelectase) des dem verengten Bronchus entsprechenden Gebietes. Zwischen dem ersten und anderen Stadium findet sich ein Übergangsstadium mit inspiratorischer Atelectase und expiratorischem Emphysem.

Die anatomische Verteilung der Bronchien bewirkt, dass die Veränderungen in geeigneten Projektionen keilförmig erscheinen. Bei den in Stadium 1 und 3 vorhandenen Veränderungen nimmt die dem verengten Bronchus entsprechende Lungenregion ein kleineres Volumen ein als normalerweise. Die Aussenlinien der keilförmigen Schatten nehmen dann oft einen konkaven Verlauf. Bei den in Stadium 2 vorhandenen Veränderungen nimmt die dem verengten Bronchus entsprechende Lungenregion ein grösseres Volumen ein als normal. Die keilförmigen aufgehellten Gebiete erhalten dann eine konvexe Begrenzung. In vorgeschrittenen Formen erhalten die Veränderungen dann eine spärische Form. Da die Volumenreduktion im Stadium 1 und 3 während der Inspiration markierter ist als bei der Exspiration, und die Volumenzunahme in Stadium 2 bei der Ausatmung grösser als bei der Einatmung, wird die respiratorische Oszillation von Herz und Mediastinum in allen Stadien vorsichgehen.

Eine Bronchostenose kann folgendermassen entstehen: durch Lumenveränderungen, Bronchialwandveränderungen und Veränderungen ausserhalb des Lumens.

Der Verf. berichtet über das Vorkommen und den Verlauf der Bronchostenose bei verschiedenen Lungenkrankheiten und bespricht die Bedeutung einer korrekten Auffassung der Radiogramme und der Komplikationen, die auftreten können.

RÉSUMÉ

La bronchosténose est un phénomène fort commun en diverses espèces de maladies pulmonaires. Au point de vue radiologique, il peut se diviser en 3 stades: Le 1:er stade présente une faible atélectasie jointe à une hyperhémie, le 2:e stade présente un emphysème local et une anémie et le 3:e stade présente un collapsus pulmonaire massif (atélectasie obstructive) de la surface correspondant à la bronche rétrécie. Entre le 1:er et le 2:e stade, il existe un stade transitoire avec atélectasie inspiratoire et emphysème expiratoire.

La distribution anatomique des bronches donne aux altérations, par projections convenables, une apparence cunéiforme. Avec les altérations se présentant dans les stades 1:er et 3:e, la région pulmonaire correspondant à la bronche rétrécie occupe un volume inférieur à la normale. Les ombres cunéiformes ont alors souvent un contour concave. Avec les altérations se présentent au 2:e stade, la région pulmonaire correspon-

dant à la bronche rétrécie occupe un volume supérieur à la normale. Les surfaces cupuliformes éclairées ont alors un contour convexe. Dans les formes avancées, les altérations prennent une configuration sphérique. Puisque la réduction de volume, aux 1:er et 3:e stades, est plus prononcée pendant l'inspiration que pendant l'expiration, et que l'accroissement de volume au 2:e stade est plus fort pendant l'expiration que pendant l'inspiration, il se produira une oscillation respiratoire du cœur et du médiastin dans tous les stades.

Une bronchosténose peut provenir des causes suivantes: altérations du lumen, altérations de la paroi bronchiale et altérations au dehors du lumen.

L'auteur donne un rapport sur la présence et le cours de la bronchosténose dans différentes formes de maladies des poumons et discuté l'importance d'une interprétation correcte des radiogrammes et des complications pouvant résulter de cette maladie.

AUS DER RÖNTGENABTEILUNG DES UNIVERSITÄTSKRANKENHAUSES IN UPSALA
(CHEF: PROF. HUGO LAURELL)

ZUR RÖNTGENOLOGISCHEN DIFFERENTIALDIAGNOSE ZWISCHEN PLEURITIS UND EMPYEM NEBST EINIGEN WICHTIGEN KLINISCHEN DATEN[1]

von

Sten Oldberg

Bei der röntgenologischen Differentialdiagnose zwischen serösem Pleuraexsudat und Empyem spielt das Verhältnis des Zwerchfells eine grosse Rolle.

Unter normalen Verhältnissen ist die Stellung des Zwerchfells in Ruhe teils durch den Druck in der Brusthöhle bestimmt, der hauptsächlich auf der Retraktionskraft der Lunge beruht, teils durch den intraabdominalen Druck, der eng mit dem Muskeltonus des Zwerchfells selbst zusammenhängt. Die Retraktionskraft der Lunge wechselt bekanntlich sowohl unter normalen als unter pathologischen Verhältnissen stark. Sie vermindert sich unter anderem, wenn sich die Lunge in ihrer Gesamtheit zusammenzieht, wie beim Ausatmen und bei Sero- oder Pneumothorax. Der intraabdominale Druck hängt nicht bloss mit dem Tonus und der Innervation des Zwerchfells zusammen, sondern auch mit der Beschaffenheit der Bauch- und Thoraxwand, ferner mit der Grösse und dem Gewicht des Bauchinhalts, sowie mit der Körperstellung. HOFBAUER und HOLZKNECHT haben klar die grosse Bedeutung der Körperlage für die Stellung des Zwerchfells nachgewiesen. Bei aufrechter Stellung sinkt das Zwerchfell im Vergleich mit liegender Stellung stark nach unten, weil das Gewicht der Eingeweide des Bauches und in gewissem Grade auch der des Thorax das Zwerchfell bei diesen Körperstellungen in verschiedener Weise beeinflusst. Bei aufrechter Stellung wird das Zwerchfell u. a. von der Leber und dem Herzen gesenkt, im Liegen dagegen steigt es vor allem wegen der veränderten statischen Druckverhältnisse im Bauch in die Höhe (siehe unten!). Eine je geringere Stütze die Bauchwand bei aufrechter Stellung den Baucheingeweiden gibt, um so mehr senkt sich das Zwerch-

[1] Bei der Red. am 28. VI. 1938 eingegangen.

fell. Gleichgewicht und Stillstand des Diaphragmas treten durch die beim Sinken wachsende Spannung der Bauchwand und der Thoraxorgane ein. Eine straffe Bauchwand hat deshalb insofern grosse Bedeutung, als sie einer starken orthostatischen Enteroptose und damit auch einer starken Diaphragmaptose entgegenwirkt. Die Beschaffenheit der Thoraxwand spielt gleichfalls eine gewisse Rolle für diese orthostatische Zwerchfellsenkung, denn wenn die Baucheingeweide die vordere Bauchwand nach vorn pressen, wird gleichzeitig deren thorakaler Ansatz nach unten gezogen, wobei die untere Thoraxappertur und damit auch das Zwerchfell sinkt. Dieser Senkung wirken die Inspirationsmuskeln und die Ligamente des Thorax entgegen.

Der Druck in der Bauchhöhle wechselt in den verschiedenen Gebieten und beruht vor allem darauf, in welcher Höhe er gemessen wird. Er variiert mit der Höhe in gleicher Weise wie der hydrostatische Druck, doch beruht er in gewissem Grade auch auf dem Spannungszustand des Zwerchfells und der Bauchwände im übrigen. Der intraabdominale Druck ist also bei aufrechter Stellung am stärksten auf dem Beckenboden und am geringsten unter den Zwerchfellkuppeln, wo er innerhalb eines grösseren oder kleineren Bereichs der subphrenischen Räume geradezu negativ ist. Das Nullniveau liegt verschieden hoch je nach der Grösse der Kraft, mit der das Zwerchfell und die übrigen Bauchwände zusammen den Bauchinhalt umschliessen, und schwankt auch in gewissem Grade mit der Grösse und Kompressibilität des Inhalts, welch letztere vor allem mit dem Luftgehalt des Ventrikels und der Därme sowie mit dem Blutgehalt der Bauchhöhle variiert. Bei Rückenlage steigen die hinteren dekliven Teile des Zwerchfells in die Höhe, weil der »hydrostatische Druck» in der Bauchhöhle beim Übergang von aufrechter Stellung zu Rückenlage in den hinteren Teile der subphrenischen Räume stark wächst. Der Druck in den vorderen Teilen der letzteren bleibt dagegen negativ. In gewissen Fällen kann es vorkommen, dass das Zwerchfell bei Rückenlage vorn etwas tiefer steht als bei aufrechter Stellung. Dies dürfte jedoch nicht nur auf dem relativ geringen subphrenischen Druck daselbst beruhen, sondern auch darauf, dass die Leber bei Rückenlage nach hinten sinkt und dass der Sinus phrenicocostalis dabei vorn mehr freigemacht wird als im Stehen, wenn die Verhältnisse im übrigen hierfür günstig sind.

Bekanntlich können pathologische Zustände in der Brust- und Bauchhöhle die Stellung und die Bewegungen der Zwerchfellkuppeln beeinflussen. Wir wollen im folgenden etwas näher darauf eingehen, wie sich ein Erguss in der Pleurahöhle dabei verhält, und besonders, *inwieweit sich die septischen Empyeme anders verhalten als gewöhnliche seröse und serofibrinöse Pleuraexsudate.*

Ein minimales, flüssiges, freies Pleuraexsudat tritt oft gar nicht aus den Lungenbildern hervor, die bei sitzender oder stehender Stellung de

Patienten aufgenommen werden. LAURELL und HJELM haben nachgewiesen, dass ein solches Exsudat durch das einfache Verfahren sichtbar gemacht werden kann, dass man den Patienten bei horizontaler Strahlenrichtung mit der kranken Seite nach unten röntgenphotographiert, wobei sich die Flüssigkeit in der Flanke als ein *Mantelexsudat* sammelt. In diesen Fällen nimmt das Zwerchfell gewöhnlich Normalstellung ein und gibt nur gelegentlich durch schlechtere Beweglichkeit beim Atmen zu erkennen, dass ein pathologischer Prozess vorliegt. Bei Ansammlung eines mässigen oder grossen freien Pleuraexsudat wiederum senkt sich das Diaphragma auf der kranken Seite, und das Mediastinum verlagert sich gleichzeitig nach der gesunden Seite. Das freie Exsudat hat gewöhnlich eine *konkave Grenze gegen die Lunge.*

Bei einem grossen rechtsseitigen Exsudat kann es schwer sein, die Stellung der rechten Zwerchfellkuppel zu beurteilen, weil der Schatten, den das Exsudat gibt, ohne sichtbare Grenze mit dem Zwerchfell-Leber-Schatten zusammenfliesst. Einen gewissen Anhaltspunkt kann man erhalten, indem man die Lage der unteren Leberfläche sowie den Grad der Verschiebung des Mediastinums beobachtet. Bei linksseitiger Pleuritis kann man die Stellung des Zwerchfells leichter beurteilen infolge des Gases, welches sich oft, besonders beim Sitzen und Stehen, direkt unter der linken Zwerchfellkuppel im Magensack und in der linken Colonflexur sammelt. Fehlen Gase in diesen Organen, so kann man solche leicht zuführen, indem man Brausepulver in den Ventrikel bringt oder Luft per rectum in das Colon bläst. Selten liegt die Milz in so grossem Umfang der linken Zwerchfellkuppel an, dass sie es ganz unmöglich macht, in dieser Weise das linke Zwerchfell oder Teile desselben zu lokalisieren.

Bei Untersuchung grosser linksseitiger Exsudate findet man in der Regel leicht, dass das linke Zwerchfell gesenkt ist. Diese Senkung ist am grössten bei aufrechter Stellung und beruht dann hauptsächlich auf der Schwere des freien Exsudats. Das Zwerchfell kann bisweilen auf der linken Seite geradezu invertiert werden. Beim Liegen, wo das Gewicht des Exsudats nicht in demselben Grade wie bei aufrechter Stellung die Lage des Zwerchfells beeinflussen kann, beruht die Senkung vor allem auf dem Seitendruck, der im Exsudat in der Richtung gegen das Zwerchfell herrscht. Dieser wechselt mit der Höhe der Exsudatsäule nach hydrostatischen Gesetzen, ist aber auch von dem Volumen des Exsudats und dem Grade der Nachgiebigkeit der es umgebenden Wände abhängig. Je mehr sich die Lunge auf der kranken Seite retrahiert hat und je mehr das Mediastinum nach der gesunden Seite verschoben worden ist, um so höher ist der Druck im Exsudat. Die Mediastinalverschiebung ihrerseits beruht nicht bloss auf der Grösse des Exsudats und dem Druck in ihm, sondern auch auf dem Retraktionsgrad und Retraktionsvermögen der *beiden* Lungen sowie auf der grösseren oder geringeren Nachgiebigkeit des

Mediastinums, alles Faktoren, die einander mehr oder minder beeinflussen.

Die Verschiebung des Mediastinums und damit auch des Centrum tendineum nach der gesunden Seite bei grossem Pleuraexsudat trägt zu ihrem Teil zu der Zwerchfellsenkung auf der kranken Seite bei, denn durch diese Seitenverschiebung des Centrum tendineum wird offenbar das Zwerchfell bis zu gewissem Grade auf der kranken Seite abgeplattet und gleichzeitig gesenkt.

Der Tonuszustand und die Kontraktionsfähigkeit des Zwerchfells auf der kranken Seite haben auch eine gewisse Bedeutung für die Stellung des Diaphragmas bei Pleuraergüssen, vor allem im Liegen, wo das Gewicht des Pleuraexsudats das Zwerchfell nicht so stark senken kann wie bei aufrechter Stellung. Dass eine Verminderung des Zwerchfelltonus eine gewisse Kephalwanderung der Zwerchfellkuppel zur Folge hat, versteht sich von selbst. Aus Beobachtungen nach Phrenicusexärese ergibt sich, dass eine vollständige Lähmung eine bedeutende derartige Kephalwanderung herbeiführt.

Eine partielle oder totale Zwerchfellähmung tritt bei basalen septischen Empyemen auf und ist eine der Ursachen der dann entstehenden Hochlage des kranken Zwerchfells. Es ist unschwer verständlich, dass dieser dünne Muskel in seiner Funktion leicht durch das toxische Exsudat geschädigt werden kann, welches sich bei septischen Empyemen bildet. Pathologisch-anatomisch hat man auch bedeutende Muskelschädigungen in Form von Myodegeneration und Rundzelleninfiltration nachweisen können. Die Lähmung des Zwerchfells auf der kranken Seite hat eine stark verminderte oder aufgehobene respiratorische Beweglichkeit zur Folge. Ein geringer Grad von Beweglichkeit kann jedoch zurückbleiben als Ausdruck der grossen respiratorischen Bewegungen der gesunden Seite, welche mittelst des Centrum tendineum vor allem die medialen Teile des kranken Zwerchfells beeinflussen.

Bei linksseitigem basalem Empyem kann man die Lage des linken Zwerchfells in der Regel leicht infolge Anwesenheit von Gas im Ventrikel und in der linken Colonflexur beobachten. Solche Gasansammlung pflegt nicht bloss bei Empyemen, sondern auch bei anderen septischen Zuständen ausgesprochener zu sein als normaliter. Möglicherweise beruht sie bei basalem Empyem auch auf der Zwerchfellähmung, die nach HITZENBERGER eine grosse Gasblase im Ventrikel hervorruft. Wenn bei linksseitigem basalem Pleuraerguss eine grosse Infektionsmilz zwischen dem Zwerchfell und den Gasblasen im Ventrikel und Colon liegt, ist zwar die Bestimmung der exakten Lage des Zwerchfells erschwert, aber die respiratorische Unbeweglichkeit, die oft bei basalem Empyem vorhanden ist, kann ebensogut an der unteren Fläche der Milz wie an der des Zwerchfells abgelesen werden.

Die Hochlage des Zwerchfells bei basalem Empyem beruht nicht bloss auf Diaphragmalähmung, denn solchenfalls müsste man erwarten, dass das Zwerchfellgewölbe, wie bei Lähmung nach Phrenicusexärese, mehr oder minder kuppelförmig ist, aber dies ist nicht der Fall. Statt dessen sieht man oft, dass das Zwerchfellgewölbe auf der kranken Seite bei aufrechter Stellung eine abschüssige Ebene bildet, die in der Flanke oder anderswo an der Thoraxwand, wo das Empyem liegt, am höchsten steht und von da mit einer ziemlich geraden Kontur nach dem Centrum tendineum abfällt. Am leichtesten beobachtet man dies, wenn das Empyem seinen Sitz in der linken Flanke hat. Die Ursachen der Aufbiegung des Zwerchfells in der Gegend der Thoraxwand oder der Wirbelsäule, wo das Empyem liegt, dürfte auch mit anderen Faktoren als der Zwerchfellähmung zusammenhängen. Dies ergibt sich aus folgendem. Die Mehrzahl der Pleuraempyeme entsteht bekanntlich auf Grundlage eines entzündlichen Prozesses in der Lunge. Meist handelt es sich um postpneumonische Empyem. Die Patienten sind nämlich gewöhnlich bettlägerig und schwer krank, schon bevor sich das Empyem entwickelt hat, wobei das Zwerchfell bei Rückenlage hinten hoch steht. Wegen Schmerz und Stechen beim Atmen sucht der Patient die kranke Thoraxhälfte möglichst zu immobilisieren. Dadurch wird die kranke Zwerchfellkuppel leicht mit der Thoraxwand in der Gegend des pneumonischen Lungenprozesses zusammengelötet, wenn letzterer — wie es die Regel ist — mit Fibrinbelag auf der Lungenfläche einhergeht. Wenn dann ein Empyem in derselben Gegend entsteht, wird das Zwerchfell örtlich gelähmt, und diese Lähmung breitet sich immer mehr aus, je grössere Teile des Diaphragmas mit dem toxischen Pleuraexsudat in Berührung kommen. Die Interposition, die so zwischen der kranken Lunge und benachbarten Thorax- und Zwerchfellteilen entsteht, bewirkt infolge der Rückenlage und des verminderten oder aufgehobenen Zwerchfelltonus, dass vor allem die Lunge sich retrahiert, wenn beim Wachsen des Empyems die Fibrinadhärenzen zwischen der Pleura visceralis einerseits und der Pleura thoracalis bzw. diaphragmatica andererseits gesprengt werden. Möglich ist, dass das Zwerchfell anfangs, statt sich zu retrahieren, in die Höhe steigt, um sich später, wenn das Empyem weiterhin an Grösse zunimmt, ein begrenztes Stück zu senken, so weit, wie die Zusammenlötung des Sinus phrenicocostalis es gestattet.

Natürlich fällt die oben beschriebene Hochlage des kranken Zwerchfells vor allem oder nur bei Untersuchung des basalen Empyems in sitzender oder stehender Stellung des Patienten in die Augen, denn die hohe Lage kontrastiert dann gegen die normale, starke Senkung des gesunden Zwerchfells. Auch nach der Heilung des Empyems bleibt das kranke Zwerchfell lange, ja oft für das ganze Leben, mehr oder minder

hoch aufgebunden, was eine herabgesetzte Zwerchfellatmung auf der kranken Seite zur Folge hat.

Auch wenn das basale Empyem nur eine mässige Grösse besitzt, wirkt es oft bei der Röntgenuntersuchung wesentlich grösser, als es in Wirklichkeit ist; besonders gilt dies für rechtsseitiges Empyem, welches gewöhnlich ohne sichtbare Grenze mit dem hochstehenden Leberschatten verschmilzt. Ausserdem ist die kranke Lunge infolge Atelektase mehr schattenbildend als normaliter und die Atelektase wegen der hohen Zwerchfellage bemerkenswert stark im Verhältnis zu der mässigen Grösse des Empyems. Eine Fehlbeurteilung der Lage des Empyems ist natürlich unter solchen Umständen bei klinischer Untersuchung noch leichter möglich als bei röntgenologischer. Die Erfahrung hat auch gelehrt, dass *die Punktion bei basalem Empyem oft zu tief vorgenommen wird*, wobei die Spritzenspitze in die Leber, die Milz oder die Bauchhöhle statt in die Empyemhöhle kommt. In einigen Fällen konnte nach solcher »Pleurapunktion» röntgenologisch freies Gas in der Bauchhöhle nachgewiesen werden. Bisweilen kommt es auch vor, dass der Chirurg, wenn er eine Empyemhöhle an ihrem tiefsten Punkt zu dränieren sucht, statt dessen die Peritonealhöhle öffnet. Wie wichtig es ist, nicht zu tief zu dränieren, zeigt auch ein Fall, den wir kürzlich beobachtet haben. Nach einer gewöhnlichen Dränageoperation mit Resektion von Costa X blieb lange Zeit nach Entfernung des Dränageröhrchens eine sezernierende Fistel zurück, und der Allgemeinzustand des Patienten besserte sich nur langsam. Bei Röntgenuntersuchung wurde festgestellt, dass das Dränageloch bedeutend tiefer lag als die in der Flanke hoch aufgebundene Zwerchfellkuppel, was die schlechte Dränage erklärte. Nach breiter Aufschlitzung der Fistel mit verbesserter Dränage wurde der Patient bald völlig gesund. Der unten mitgeteilte Fall Nr. 2 gehört nicht zu den Seltenheiten, und sein Röntgenbild zeigt, dass die Probepunktion oft hoch oben in der Axille ausgeführt werden muss, damit man die Empyemhöhle erreicht. Beachtet man dies nicht, so besteht immer die Gefahr, dass ein basales Empyem übersehen oder seine rationelle Behandlung unnötig aufgeschoben wird.

Von Interesse ist, dass sich das Mediastinum während des Atmens bei grossen basalen Pleuraexsudaten und basalen septischen Empyemen verschieden verhält (LAURELL). Bei den serösen oder serofibrinösen Pleuraexsudaten pendelt das Mediastinum während des Atmens hin und her, inspiratorisch nach der Pleuritisseite, exspiratorisch in entgegengesetzter Richtung. Dieses Pendeln beruht vor allem darauf, dass das Zwerchfell auf der kranken Seite stark gesenkt und abgeplattet ist, wodurch bei seiner Kontraktion eine grosse Kraftresultante das Centrum tendineum nach der kranken Seite hinüberzieht. Dazu kommt, dass die entsprechende Kraftresultante auf der gesunden Seite kleiner ist als

normal infolge der durch die exspiratorische Verschiebung des Mediastinums nach dieser Seite verstärkten Kuppelbildung des Zwerchfells daselbst. Das respiratorische Pendeln bei Pleuritis kann eine Grösse von 2—3 cm und noch mehr erreichen. Es erinnert an das Pendeln bei einseitiger Bronchostenose und Pneumothorax, ist aber in der Regel kleiner als bei diesen Zuständen. Andere Voraussetzungen für ausgesprochenes Pendeln bei grossem Pleuraexsudat sind, dass der Thorax der kranken Seite wesentlich an der Respiration teilnimmt sowie dass die Lunge auf der kranken Seite so zusammengefallen und atelektatisch ist, dass sie wenig oder gar nicht inspiratorisch erweitert wird; bekanntlich leistet eine atelektatische Lunge, im Gegensatz zu einer mässig luftgefüllten, dem Zutritt der Luft starken Widerstand. Unter den genannten Voraussetzungen tritt eine ausgesprochene inspiratorische Drucksenkung im Pleuraexsudat ein, und eine solche ist notwendig, damit die gesenkte Zwerchfellkuppel auf der kranken Seite inspiratorisch das Centrum tendineum mit dem unteren Mediastinum nach dieser Seite hinüberziehen kann. Denn sinkt nicht der Druck im Pleuraexsudat während der ganzen Phase des Einatmens durch eine fortschreitende Thoraxerweiterung, so steigt der Druck im Exsudat rasch bei dem Streben des Zwerchfells, das Mediastinum hinüberzuziehen, wobei das respiratorische Pendeln gehemmt wird und rasch aufhört.

Durch die Lähmung des Zwerchfells *bei basalem Empyem nimmt das respiratorische Mediastinalpendeln stark ab*, ein oft wichtiges differentialdiagnostisches Symptom. Die Ursache hiervon ist jedoch nicht nur die Lähmung, sondern auch die herabgesetzte thorakale Atmung auf der kranken Seite. Diese Herabsetzung beruht teilweise auf Stechen und Schmerz beim Atmen, teilweise auch auf dicken Fibrinpelzen, welche die Empyemhöhle auskleiden und die kranke Lunge an die Thoraxwand binden. Durch die verminderte Thoraxatmung auf der kranken Seite nimmt auch die inspiratorische Drucksenkung bzw. die exspiratorische Drucksteigerung daselbst ab, womit auch eine wichtige Voraussetzung für grosses respiratorisches Pendeln fortfällt. Die genannten Schwarten um die Empyemhöhle sind bei der Operation insofern von Vorteil, als sie bei der Thorakotomie eine starke Mediastinalverschiebung und andere schwere Komplikationen hindern, die bei einem offenen Pneumothorax auftreten können.

Wenn man von respiratorischem Pendeln spricht, darf man nicht vergessen, dass auch ein normales, wenngleich ziemlich unbedeutendes solches Pendeln vorkommt, welches vor allem auf verschiedener Blutfüllung der Herzräume während des Atmens sowie auf der exzentrischen Lage des Herzens beruht (LAURELL). Das Herz pendelt nämlich oft einige Millimeter bis ca. 1 cm hin und her, inspiratorisch nach rechts, exspiratorisch nach links. Ebenso muss man sich daran erinnern, dass

alles, was die Erweiterung der Lunge vor allem in seitlicher Richtung hindert, ein respiratorisches Pendeln nach der kranken Seite hervorrufen kann, also alle krankhaften Veränderungen in Lunge und Pleura, welche die Dehnbarkeit der Lunge herabsetzen.

Noch ein weiteres Symptom von Pleuraempyem ist besonders hervorzuheben; dasselbe ist wichtig, weil es auftreten kann, wo auch immer der Erguss lokalisiert ist. Die Pleuraempyeme zeigen nämlich in der Regel eine grosse Tendenz zu Abkapselung und haben dann eine *konvexe Grenze gegen die Lunge*, während ein freies Pleuraexsudat eine konkave solche Begrenzung hat. Der subpleurale Abszess dagegen hat, wenn er der Thoraxwand anliegt, zentral eine konvexe, aber auf beiden Seiten davon eine konkave Grenzlinie gegen die Lunge, wie Beobachtungen in der hiesigen Abteilung gezeigt haben (SKARBY). Ein grosses sekundärinfiziertes Pleuraexsudat hat gewöhnlich keine konvexe Grenze gegen die Lunge.

Bei der röntgenologischen Differentialdiagnose zwischen Empyem und Pleuritis ist also — abgesehen von wichtigen klinischen Symptomen — zu beachten, ob der Erguss in der Pleura frei oder abgekapselt ist, ferner, wenn der Erguss bis zum Zwerchfell hinabreicht, dessen Lage im Sitzen und seine respiratorischen Verhältnisse sowie endlich die Lage und das Verhalten des Mediastinums beim Atmen.

In die nachstehende Kasuistik sind nur einige lehrreiche Fälle aufgenommen.

Fall 1. E. J. 9 Jahre. Pat. erkrankte am 4. 5. 1937 mit Zeichen von rechtsseitiger Oberlappenpneumonie. Die Temperatur sank in gewöhnlicher Weise kritisch und war am 15. 5. afebril. Am 22. 5. plötzlich Temperaturerhöhung auf 39°. Pat. wurde am 27. 5. in die hiesige Pädiatrische Abteilung aufgenommen, war damals ziemlich mitgenommen und wies physikalische Zeichen von rechtsseitigem Pleuraexsudat auf. Das Röntgenbild zeigte eine Verschleierung über dem ganzen rechten Lungenfeld, die nach der Basis zunahm, sowie einen dünnen Mantelbelag mit interlobärer Fortsetzung. Die rechte Zwerchfellkuppel stand hoch, speziell hinten, und wies keine deutlichen Exkursionen beim Atmen auf. Die röntgenologische Diagnose lautete: perihiläre Infiltrate mit einer mässigen Menge infiziertem Pleuraexsudat auf der rechten Seite. Probepunktion der rechten Pleurahöhle in I_7 in der hinteren Axillarlinie ergab nur ein paar Tropfen trübes Exsudat, welches mikroskopisch zahlreiche polynukleäre Leukozyten und Kokken in Ketten enthielt. Erneute Pleurapunktion in I_8 in der Skapularlinie lieferte ein paar ccm trübe, graugelbe Flüssigkeit. Ein Röntgenbild vom 29. 6. zeigte eine unbedeutende Verschleierung über den unteren Teilen des rechten Lungenfeldes und, wie vorher, ein hohes, gewölbtes Zwerchfell. Am 10. 7. wurde Pat. symptomfrei entlassen.

Fall 2. K. B. 51 Jahre. Pat. erkrankte am 13. 1. 1937 an typischer Pneumonie. Ein paar Wochen nach der Erkrankung war die Temperatur noch immer subfebril, und nach etwa einer weiteren Woche stieg sie von neuem. Das Röntgenbild am 12. 3. zeigte einen in der rechten Flanke gelegenen, sich gegen das Lungenparenchym vorwölbenden, dichten Schatten sowie eine an diesem hoch aufgebundene Zwerchfellkuppe (Fig. 1). Diagnose: abgekapseltes Empyem. Probepunktion in I_7 in der rechten Axil-

Fig. 1.

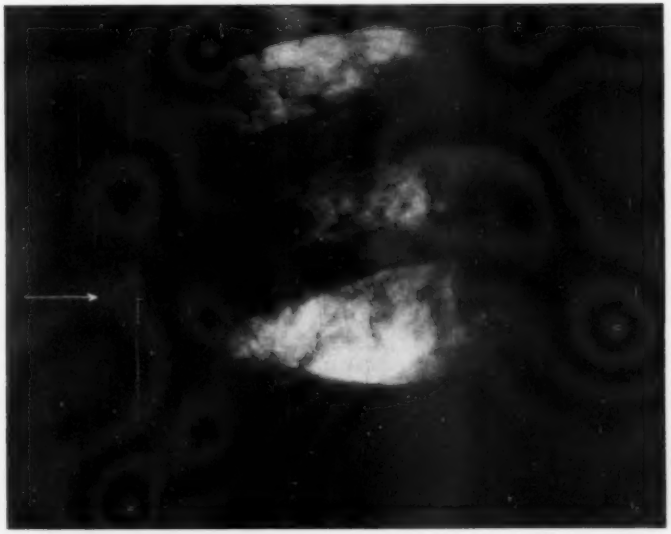

Fig. 2. Beim Pfeile horisontales Flüssigkeitsniveau.

ergab ca. 5 ccm dicken, gelbbraunen Eiter mit Detritus, Leukozyten und Gram-positiv[e] Diplokokken. Das Röntgenbild am 16. 2 liess erkennen, dass der sich vorwölbende Sch[at]ten in der rechten Flanke etwas an Grösse abgenommen hatte. Im Empyem konnte [ein] horizontales Niveau und oberhalb desselben ein knapp haselnussgrosser, luftiger Ho[hl]raum (Fig. 2) unterschieden werden (Luft bei der Punktion eingeführt). Am 23. 3. wur[de] Pat. subjektiv symptomfrei entlassen.

Fall 3. A. B. 40 Jahre. Pat. erkrankte am 6. 8. 1937 subakut mit Stechen [in] der rechten Brustseite, Schüttelfrösten und Temperatursteigerung auf etwa 39°. Rön[t]genuntersuchung am 17. 8 zeigte im unteren Teil des rechten Lungenfeldes einen kom[p]pakten, sich gegen das Lungenparenchym vorwölbenden Schatten, der nach oben [in] einem dünnen Mantelbelag fortsetzte. Aus dem Seitenbild ging hervor, dass der Scha[t]ten an der vorderen Thoraxwand lag und dass die rechte Zwerchfellkuppel vorn und [in] der Flanke aufgebunden war. Bei Durchleuchtung wurde eine deutliche Mediastinal[-]wanderung beim Atmen festgestellt, was also gegen Diaphragmaparese sprach. Diagnose: ein gegen die vordere Thoraxwand abgekapseltes Pleuraexsudat, wahrscheinlich seröse. Am 19. 8. wurde Probepunktion mit Hilfe eines bei Röntgendurchleuchtung aufgesetzten Indikators vorgenommen, wobei man ein grüngelbes, klares Exsudat mit einer mässigen Menge mononukleärer Leukozyten und ohne Bakterien im Direktpräparat erhielt.

ZUSAMMENFASSUNG

Verf. erörtert die Faktoren, die unter normalen Verhältnissen auf die Stellung des Zwerchfells einwirken und geht sodann auf das Röntgenbild bei Pleuraexsudat über. Durch eine genügend grosse Menge von serösem Exsudat werden die Zwerchfellkuppeln abgeflacht und gesenkt, ferner wird die respiratorische Wanderung des Mediastinums vergrössert. Bei basalen septischen Empyemen dagegen ist das Zwerchfell gewöhnlich hoch an die Brustkorbwand hinaufverlagert und mit ihr verwachsen. Dies ist zum grossen Teil durch eine Zwerchfellähmung bedingt, die auch eine bedeutende Abnahme der Mediastinalwanderung mitsichbringt. Seröse Exsudate haben meistens eine konkave Grenze gegen die Lunge, die Empyeme dagegen werden abgekapselt und weisen eine gegen das Lungenparenchym gebuchtete Kontur auf. Verf. hebt die Wichtigkeit hoher Punktion bei Verdacht auf Empyem hervor. Zum Schluss ein Bericht über drei instruktive Fälle.

SUMMMARY

The author discusses first the factors which normally influence the position of the diaphragm and then passes on to the roentgen picture of pleural exudate. A sufficiently large quantity of serous exudate flattens and depresses the cupola of the diaphragm and increases the respiratory movement of the mediastinum. In basal septic empyema on the other hand, the diaphragm is usually held up high against the chestwall. This is due chiefly to a paresis of the diaphragm which also considerably reduces the movement of the mediastinum. Serous exudate usually shows a concave border towards the lung while empyema becomes encapsulated and presents a bulging contour toward the pulmonary parenchymæ. The author points out the importance of high paracentesis when empyema is suspected and gives an account of three illustrative cases.

RÉSUMÉ

L'auteur discute tout d'abord les facteurs qui, par circonstances normales, influent sur la position du diaphragme, puis passe au radiogramme d'exsudations de la plèvre.

Une quantité suffisamment grande d'exsudations séreuses provoque un aplatissement et un affaissement des coupoles du diaphragme, et augmente le mouvement respiratoire du médiastin. Dans les empyèmes septiques basals, par contre, le diaphragme est généralement fortement relevé contre la paroi thoracique. Ceci dépend en grande partie d'une parésie du diaphragme, qui amène également une diminution considérable du mouvement du médiastin. Les exsudations séreuses présentent le plus souvent une limite concave vers le poumon, tandis que les empyèmes, par contre, deviennent encapsulés et présentent des contours convexes vers le parenchyme pulmonaire. L'auteur fait ressortir l'importance de hautes ponctions lorsqu'est suspecté un empyème. Il relate finalement trois cas démonstratifs.

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AUS DEM RÖNTGENINSTITUT (PROF. M. LÜDIN) DER UNIVERSITÄT BASEL

REGIONÄRER GASTROSPASMUS BEI PANKREASER-KRANKUNG[1]

(Entzündung und Lithiasis)

von

Max Lüdin

Die krampfhafte Kontraktion einer umschriebenen Magenpartie kann nicht nur ausgelöst werden durch eine Wandveränderung im entsprechenden Magenabschnitt, sondern sie ist nicht selten — besonders als regionärer Gastrospasmus der präpylorischen Partie — das Zeichen eines schmerzhaften, entzündlichen Reizzustandes in einem benachbarten Organ, ein Fernsymptom, das — allgemein als viscero — visceraler Reflex nach BERGMANN aufgefasst — besonders bei Cholelithiasis, seltener auch bei Nierenstein, Appendicitis, Ulcus duodeni beobachtet worden ist.

Es war a priori zu erwarten, dass analog den genannten Affektionen auch die entzündlichen, schmerzhaften Veränderungen in einem anderen benachbarten Organ, der Bauchspeicheldrüse, in gleicher Weise krampferzeugend auf den Magen einwirken können. In der Literatur habe ich jedoch eine dahin gehende Angabe nicht finden können und möchte deshalb im folgenden kurz über 2 Fälle von regionärem Spasmus der präpylorischen Magenpartie bei Pankreassteinen und bei Pankreatitis berichten, deren Krankengeschichte resp. Sektionsbefund ich den Herren Prof. RUDOLF STAEHELIN (Medizinische Klinik), Prof. K. HENSCHEN (Chirurgische Klinik), Prof. A. WERTHEMANN (Pathologisch-Anatomische Anstalt) verdanke.

Fall 1: 66 Jahre alter Weichenwärter B. wird von der chirurgischen Klinik wegen Sigmoidtumor zur Röntgenuntersuchung überwiesen. Auszug aus der Krankengeschichte: In der Anamnese keine Angaben, die auf ein Magenleiden oder eine Pankreasaffektion hindeuten. Starke Gewichtsabnahme. Aus dem Status: Abdomen mit schlaffer Bauchmuskulatur. Im Oberbauch bei tiefer Palpation keine Resistenz fühlbar, kein Druckschmerz feststellbar. Urin: Zucker positiv 3.3 %.

[1] Bei der Redaktion am 5. VIII. 1938 eingegangen.

D*Röntgenuntersuchung* ergib einen erweiterten Mage. Die präpylorische Parti ist auffallend eng, stark kontrahiert. Der geradlin g begrenzte Schatten diese Magenpartie zieht zapfe förmig gegen das Duod num hin. An der unteren echten Ecke des L.W. I m tiple Pankreassteinschat n (Abb. 1). Bei der Schrä aufnahme zeigt sich dieselbe Formation der präpylorischen Partie mit scharfer Absetzung gegen den übrigen Magenschatten zu (Abb. 2). Eine spätere Wiederholung der Aufnahme zeigt die präpylorische Partie wieder scharf abgesetzt gegen den Magenkörper, sie ist kaum gefüllt, lässt nur einige geradlinig verlaufende Schleimhautfalten erkennen (Abb. 3). Die Röntgenuntersuchung ergibt ferner eine Pylorusverschieblichkeit von einer Wirbelhöhe. Der Magen zeigt nach 6 Stunden noch schwachen Schleimhautbelag.

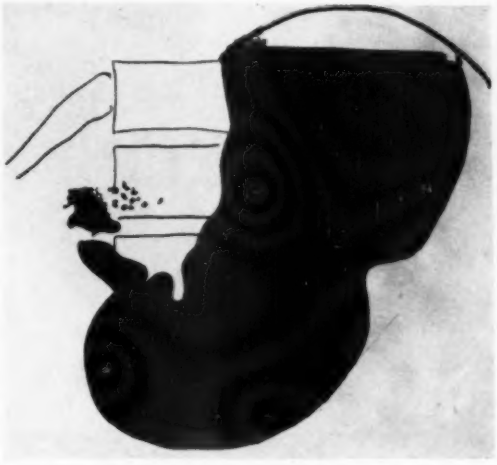

Abb. 1. Pankreassteine. Regionärer Gastrospasmus.

Röntgendiagnose: Pankreassteine. Regionärer Gastrospasmus der präpylorischen Partie.

Auszug aus dem *Sektionsprotokoll:* Der Pylorus ist stark hypertrophisch, der Magen leicht gefältelt, der Antrumteil grau pigmentiert. Das Pankreas ist ziemlich schmal und ausserordentlich fest. Auf Schnitt zeigt sich, dass Drüsengewebe kaum mehr vorhanden ist, dagegen ist es durch Bindegewebszüge ersetzt, die ziemlich weisslich glänzendes Aussehen aufweisen. Die Drüsengänge sind ziemlich stark erweitert. Im Verlaufe des Pankreasganges werden eine grosse Anzahl weisslicher, glänzender, griesförmiger Konkremente angetroffen, die zum grossen Teil miteinander verbackene Konkremente von über Linsengrösse bilden und den Ausführungsgang

Abb. 2. Schrägaufnahme zu Abb. 1. Pankreassteine Regionärer Gastrospasmus.

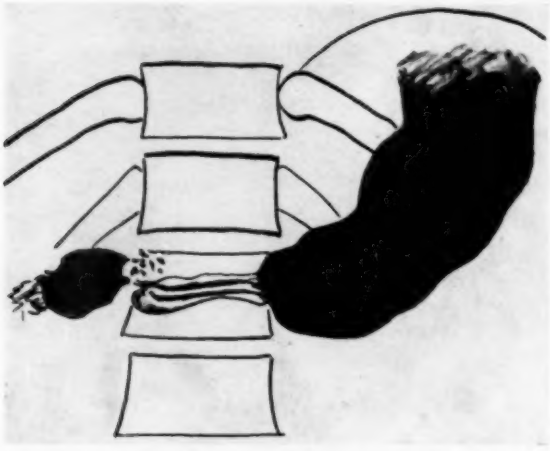

Abb. 3. Kontrollaufnahme zu Abb. 1. Pankreassteine Regionärer Gastrospasmus.

stellenweise verlegen. Die Wand des Ganges ist verdickt. Das Lumen besonders in der Gegend der Papille stark erweitert. Mikroskopische Untersuchung des Pankreas: Pankreas frei von entzündlichen Infiltraten, dagegen etwas bindegewebig sklerosiert. Im Kopfteil findet sich schwerste bindegewebige Verödung und Hyperplasie. Die Drüsenläppchen sind ganz atrophisch und von neu gebildetem Bindegewebe diffus durchsetzt.

Epikrise: Bei einem Patienten mit Pankreassteinen, mit bindegewebiger Verödung und Hyperplasie des Pankreaskopfes und mit hochgradiger Atrophie des exkretorischen Teiles des Pankreas wird röntgenologisch am Magen ein regionärer Spasmus der präpylorischen Partie festgestellt.

Fall 2: 41 jähriger Patient Ph. Auszug aus der Krankengeschichte der medizinischen Klinik (640/37): Familienanamnese ohne Besonderheiten. Nach einem Diätfehler (Bier auf Aepfel getrunken) treten bei dem Patienten Krämpfe im ganzen Leib auf, Erbrechen. Aus dem Status: Abdomen aufgetrieben, gespannt. Keine lokale Défense. Keine Resistenz palpabel. Ganzes Abdomen bei Berührung druckempfindlich, maximal 1—2 cm über dem Nabel. Geringer Entlastungsschmerz. Auskultatorisch reichlich Gurren und tiefe, metallisch klingende Darmgeräusche. Keine Darmsteifung sichtbar. Kein Ascites nachweisbar. Temperatur 37.6°. Leukozyten 15,300. Senkungsreaktion 52/110. Urin: Zucker positiv 1.9 %. Blutzucker 0.2 %. Diastase im Serum nach Wohlgemuth 64. Diagnose: Pankreatitis akuta.

Abb. 4. Regionärer Gastrospasmus. Ausweitung des Duodenalbogens bei akuter Pankreatitis.

Die *Röntgenuntersuchung* 9 Tage nach Spitaleintritt ergibt:

Thorax: Hochstehendes, nur mässig bewegliches, linkes Zwerchfell. Sinus phrenicocostalis beidseits frei. Schräg verlaufender Schattenstreifen an der linken Lungenbasis (Plattenatelektase). Magen: Der Kontrastschatten des normalen Magenkörpers ist beim Übergang zur präpylorischen Partie scharf abgesetzt und geht in einen zapfenartigen, nur wenig gefüllten, stark kontrahierten präpylorischen Anteil über, in welchem normal verlaufende Schleimhautfalten eben noch erkennbar sind. Der Bulbus duodeni etwas erweitert. Der Duodenalbogen deutlich ausgeweitet. Das absteigende Duodenum erweitert (Abb. 4).

Röntgendiagnose: Regionärer Gastrospasmus der präpylorischen Partie. Erweiterung des Duodenalbogens. Vergrösserung des Pankreaskopfes? Bewegungsmangel des linken Zwerchfelles. Plattenatelektase an der linken Lungenbasis.

Epikrise: Bei einem Patienten mit akuter Pankreatitis zeigt die Röntgenuntersuchung einen regionären Gastrospasmus der präpylorischen Partie; ausserdem eine mangelhafte Beweglichkeit des linken Zwerchfelles mit darüber lokalisierter Plattenatelektase.

Aus diesen zwei Beobachtungen geht hervor, dass wir unter die bereits bekannten indirekten und funktionellen Zeichen der entzündlichen Veränderung des Pankreaskopfes als neues Symptom den regionären Gastrospasmus der präpylorischen Partie einreihen dürfen.

ZUSAMMENFASSUNG

Verfasser berichtet über einen Fall von chronischer Veränderung im Pankreaskopf mit Pankreassteinen und über einen Fall von akuter Pankreatitis. Bei beiden Fällen wurde durch die Röntgenuntersuchung ein regionärer Gastro-Spasmus der präpylorischen Partie festgestellt.

SUMMARY

The author reports a case of chronic lesion in the head of the pancreas with pancreatic concrements and also a case of acute pancreatitis. In both cases the roentgen examination made possible a diagnosis of regional gastrospasm of the præpyloric portion.

RÉSUMÉ

L'auteur rapporte un cas de lésion chronique de la tête du pancréas avec concrétions pancréatiques, ainsi qu'un cas de pancréatite aiguë. Dans les deux cas, l'examen roentgen permit de diagnostiquer un gastro-spasme régional de la portion pré-pylorique.

LES CARACTÉRISTIQUES RADIOLOGIQUES DU SOI-DISANT ULCÈRE DUODÉNAL GROS[1]

par

D. S. Bartstra

Radiologue à Leyde

En général, la grandeur de l'ulcère duodénal est donnée comme variable entre celle d'un grain d'orge et celle d'une fève.

Le soi-disant ulcère duodénal gros, au contraire, pourrait avoir la grandeur d'une noix.

Quelque improbable que cela puisse être, il paraît tout de même que justement dans ces cas-là, la niche n'est souvent pas reconnue comme tel radiologiquement.

C'est ainsi que BRDICZKA parle de trois cas; dans 2 de ces cas on n'a pas reconnue la niche. KNUTSSON donne 4 cas, dont 2 furent parfaitement bien reconnus. Finalement notre cas à nous: ici encore la niche n'a pas été reconnue comme tel. Un des cas de KNUTSSON se trouvait être un ulcère cancéreux.

Si nous avons affaire dans ces cas à un bulbe duodénal déformé, alors ce symptôme indirect peut nous rappeler l'ulcère duodénal. Il va de soi qu'on cherchera alors plutôt une niche dans le bulbe duodénal déformé. Mais encore dans ces cas-là l'expérience prouve que la niche est difficile à démontrer.

Il est encore plus difficile de reconnaître la niche quand celle-ci a la forme et la grandeur du bulbe duodénal. BRDICZKA comme KNUTSSON parlent, chacun de son côté, d'un tel exemple et notre cas à nous s'y joint. (Dans le 2e cas de BRDICZKA, il est vrai, la niche a été également prise pour le bulbe duodénal mais ce cas n'est pas trop clair, puisque nous n'y voyons pas de belle formation de bulbe duodénal.).

Avant de décrire les symptômes de l'ulcère duodénal gros, il est peut-être bon de communiquer notre cas.

[1] Remis à la Rédaction le 27 Avril 1938.

Le malade était un homme très corpulent. On nous l'envoya pour un examen radiologique de l'estomac, à cause de douleurs épigastriques à droite dont il se plaignait.

A notre examen (9. 4. 35), produisant une photographie radiologique très indistincte de ce malade corpulent, nous trouvâmes un bulbe duodénal tourné un peu en arrière, de bonne forme. Bien que cette forme montrât quelques petits changements, le bulbe duodénal pourtant ne se vidait jamais complètement. Il était dommage que nous ne pussions examiner le bulbe duodénal que dans une seule direction. Ce qui frappait le plus cependant, c'était le rétrécissement en forme de tube du duodénum, tout juste le bulbe duodénal passé, ce que nous devions attribuer à un spasme puisque cette image n'était pas constante (fig. I).

Nous crûmes pouvoir exclure un ulcère duodénal et nous indiquâmes la possibilité d'une anomalie de la vésicule biliaire avec spasme du duodénum.

A l'opération (Dr. NIJST) on trouva un très gros ulcère à la face antérieure du duodénum, tout juste le pylore passé, à la grandeur d'un bout de pouce. Il faut donc que nous avons pris ici la niche pour le bulbe duodénal.

Si nous faisons attention aux symptômes radiologiques de l'ulcère duodénal gros (nous indiquons en particulier ces gros ulcéres qui ont la forme du bulbe duodénal), alors il y a certainement des caractéristiques qui puissent nous montrer la bonne voie.

En premier lieu, la niche donne une image assez fixe et constante, C'est la raison que le bulbe est constamment rempli d'une matière peu changeante. La délimitation souple du bulbe duodénal normal manque de même que sa péristaltique physiologique, par laquelle de temps en temps le bulbe duodénal se vide et ce n'est que la reproduction de la muqueuse qui puisse rester.

Cette grosse niche ne se vide pas non plus en le comprimant contraire au bulbe duodénal normal. C'est uniquement en changeant de position que la grosse niche pourrait se vider.

En second lieu le rétrécissement de la partie supérieure du duodénum, tout juste le bulbe duodénal passé, est un symptôme de haute importance. Il faut l'expliquer principalement par spasme, mais une infiltration de cette région est encore possible.

Si nous comparons notre cas aux cas de BRDICZKA et de KNUTSSON où la grosse niche ressemble au bulbe duodénal normal, une différence nous frappe. Dans les 2 cas publiés déjà, la partie supérieure duodénale rétrécie sort tout à coup d'un bulbe duodénal dont les limites sont de forme convexe (fig. II et III). Dans notre cas à nous le bulbe duodénal passe insensiblement à la partie supérieure duodénale rétrécie (fig. I).

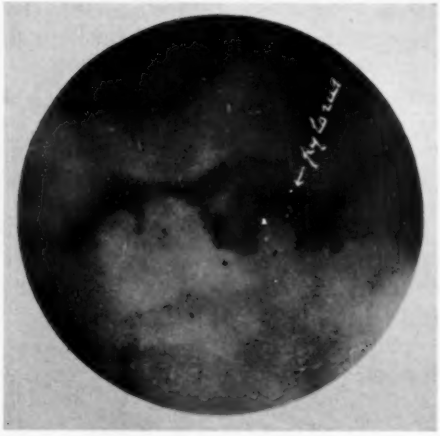

Fig. I.

Fig. II. Cas de Brdiczka.

Fig. III. Cas de Knutsson.

Finissons en signalant que les symptômes qui donnent le soi-disant ulcère duodénal gros, dépendent de la grandeur du bulbe duodénal. A savoir: si le bulbe duodénal est grand lui aussi, un gros ulcère duodénal qui s'y trouve, peut donner les mêmes phénomènes connus qui se présentent avec l'ulcère duodénal ordinaire. (Voir fig. IV et V). Afin qu'une bonne comparaison soit possible, les fig. I, IV et V ont été reproduites

Fig. IV.

Fig. V.

à grandeur naturelle. Les caractéristiques radiologiques de l'ulcère duodénal gros ne sont donc pas déterminées en premier lieu par la grandeur véritable de l'ulcère, mais plutôt par le rapport entre la grandeur de la niche et celle du bulbe duodénal.

RÉSUMÉ

Description des symptômes radiologiques de l'ulcère duodénal gros.

SUMMARY

Description of the röntgenological signs of the large duodenal ulcer.

ZUSAMMENFASSUNG

Beschreibung der röntgenologischen Erscheinungen des grossen Ulcus duodeni.

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CHIEF: NILS WESTERMARK, M. D.

ON THE ROENTGEN DIAGNOSIS OF LUNG EMBOLISM[1]

by

Nils Westermark

Brief review of the incidence, pathology and clinical symptoms of lung embolism

The acute obstructive embolus in the pulmonary artery is a well known and dreaded complication after many diseases as well as after operations. The clinical picture of this lungembolism, which in a short time ends fatally, is well known. These cases are very seldom if ever roentgenexamined.

The acute embolus in one of the main branches of the pulmonary artery or in one or several of the smaller branches in the lung, may also appear acute as a complication after many diseased conditions as well as after operations. The patient is generally taken acutely ill with sharp pains in the chest, a feeling of suffocation and a bloody sputum. The condition may also however take an insiduous onset without any such symptoms that permit a clinical diagnosis being made. It may also take on a chronic course, and may then readily be overlooked. It has not created sufficient attention from either clinical or roentgenological point of view and often ends fatally.

Minor emboli of this nature are of very frequent occurrence and are often encountered on autopsic examinations without having been clinically diagnosed. POUL MÖLLER who has devoted a thorough study to this question found among 176 autopsies lung emboli in no less than 50 cases or in about 28 %. Among these cases of lung embolism there were 10 with larger emboli with a rapidly fatal issue. In 17 cases emboli were of medium size in one of the main branches of the pulmonary artery or in the intrapulmonary branches of the first degree. In 23 cases there were minor emboli located in the smaller branches of the pulmonary artery. It is being stated by WAHLGREN who has also devoted much attention to

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this question that the minor emboli occur much more frequently tha one might have reason to expect and that they are very often found o post mortem examinations, if only carefully looked for, especially i patients who have died of some chronic disease.

MÖLLER has found that lung embolism occurs nearly twice as ofte in women as in men. The large emboli seem to subside more often i men than women without leading to death.

They occur more frequently in older than younger patients. The greatest incidence is found in the fifth to the seventh decade.

According to investigations carried out by PANNUM the cause of death is in most cases cerebral anaemia occasioned by the lung embolism. In MÖLLER's opinion, however, we have to remember, particularly in emboli of smaller or medium size, that lack of oxygen in the blood and possibly direct suffocation may also be the cause of death.

Emboli give rise to haemorrhagic infarcts of the lung only or practically only in the case of manifest stasis of the pulmonary veins, thus in cardiac weakness or in defects of the mitral valve.

In a cut section of the lung the haemorrhagic infarct has the shape of a wedge with the apex pointing towards the hilum and the base towards the pleura. The infarcted area is devoid of air.

In most cases of lung embolism, however, there is no infarct present. On the other hand if there is no stasis of the pulmonary circulation we get ischaemia of that part of the lung corresponding to the distributive area of the embolic branch of the pulmonary artery. Since the lung area is nourished by branches of the bronchial artery an ischaemic infarct can only arise in the case of cessation of circulation in these branches. The ischaemic lung area is thus alive although no gas exchange would seem likely to take place therein.

In lung embolism without infarction the embolic lung area appears anaemic and pale besides being aerated. It is wedge-shaped, the apex being directed towards the hilum and the base towards the pleura.

In some cases of emboli of smaller or medium size we sometimes get secondary thrombosis arising through thrombotic masses being deposited on the embolus. This thrombosis may become quite extensive and even gradually cause complete occlusion of the main branch of the pulmonary artery. MÖLLER found secondary thrombosis in 50 % of his cases. In about one-fifth of the cases did the thrombosis show any larger distribution.

The clinical symptoms in non-obstructive cases of embolism are increasing breathlessness, cyanosis, anxiety, feeble pulse, pains in the chest (stitches) and a sanguinolent sputum. Clinical symptoms were present in but one-third of MÖLLER's cases of small non-obstructive embolism. The commonest and most definite symptoms were stitches

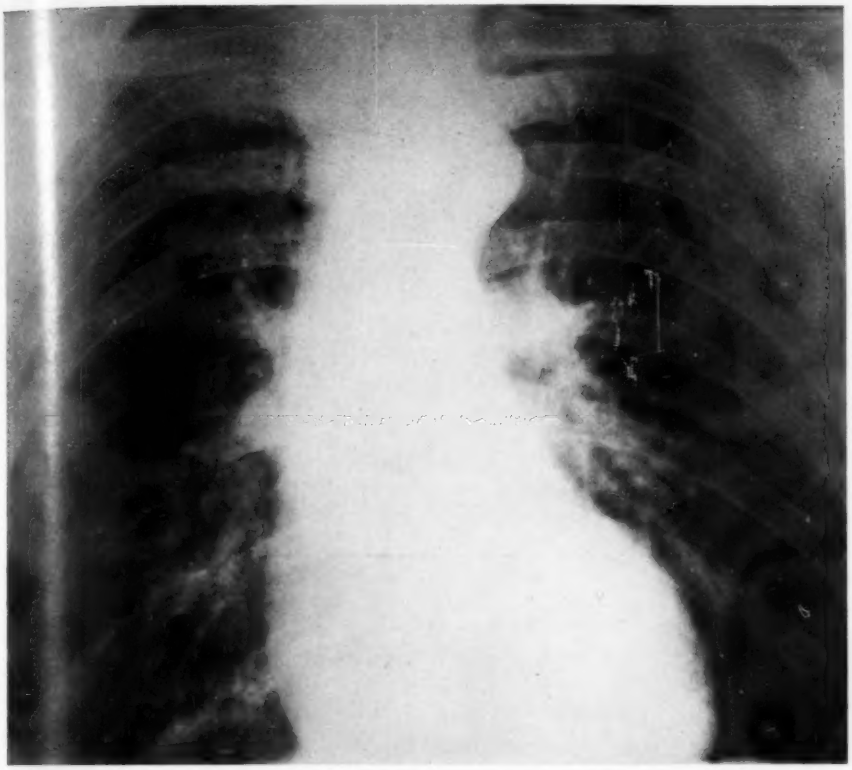


Fig. 1. Case 1. Before onset of lung embolism. There is mild hypertrophy of left side of the heart and some old age aortitis. Normal vascularisation of lungs.

and sanguinolent expectoration. There is thus a very large number of cases with no clinical symptoms whatever.

Earlier investigations into the roentgen appearance of lung embolism

According to KOHLMANN a lung infarct appears on an antero-posterior radiogram as a triangular or a rounded oblong massive shadow. In his large handbook ASSMANN writes as follows on this subject: »Der Lungeninfarkt bewirkt infolge der vollkommenen Aufhebung des Luftgehaltes in dem infarzierten Bezirk eine totale, gänzlich strukturlose Verschattung im Röntgenbilde. Entsprechend der bekannten anatomischen Keilform des Infarkts entsteht dort, wo die Achse des Keils in annähernd frontaler

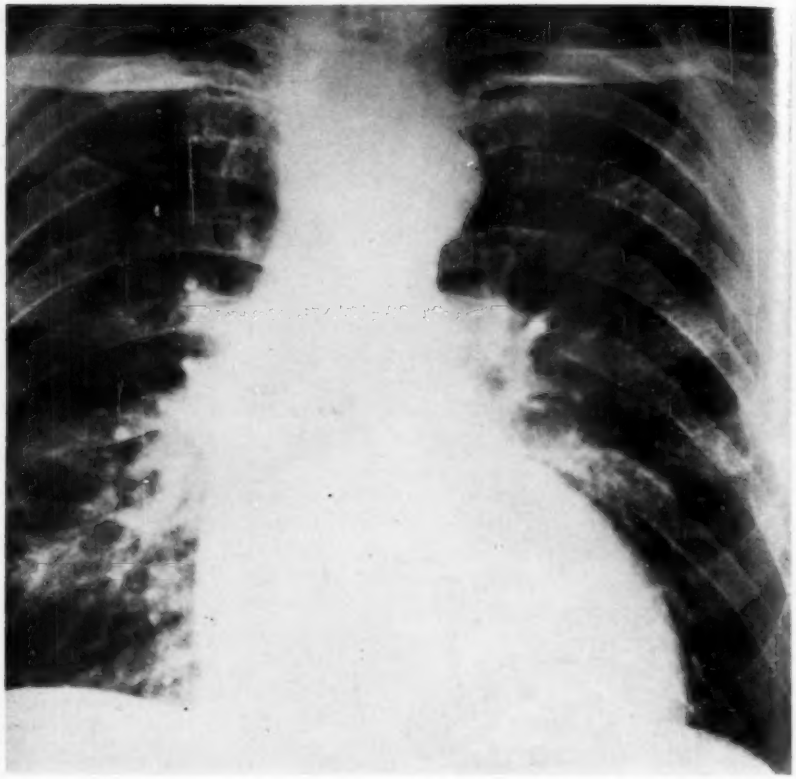


Fig. 2. Same case after onset of lung embolism. Examination varied out on day of death. Cardiac condition seems on the whole unaltered. Obvious anaemia within entire left lung and right upper lobe. The vascular design terminates abruptly in the central part of the areas corresponding to the anaemic portions.

Richtung gelegen ist, ein dreieckiger Schatten, dessen Spitze hiluswärts gerichtet ist und dessen Basis an der Thoraxwand liegt. Fällt die Achse des Keils dagegen in einen sagittalen Durchmesser, so erzeugt die rundliche Basis des Keils einen kreisrunden oder ovalären Schatten, der mehr in die Mitte des Lungenfeldes hineinprojiziert wird.» FRIMANN-DAHL dealing with postoperative lung embolism from a radiological viewpoint writes as follows regarding roentgen diagnostics: »Kleine Embolien mit schwacher Hämoptysis erzeugen flüchtige Veränderungen, ohne deutliche Infarkte zu bilden, wahrscheinlich bilden sich nur lokale Hyperämien aus. Grössere Embolien geben Veränderungen von längerer Dauer, bis zu

a. b.

Fig. 3. Case 2. Examination carried out on day before death after taken ill with pulmonary embolism. Decided anaemia of the entire right lung, particularly well marked in its upper lobe and in left upper lobe. The vascular design seems to end abruptly in the central parts of the lung.

mehreren Wochen. Auf den Röntgenbildern sieht man diese als dreieckige oder runde, stark gesättigte, charakteristische Schatten, welche gewöhnlich an der Basis der Lunge lokaliziert sind.» LIEBMANN maintains that it is difficult or impossible in most cases to distinguish the haemorrhagic lung infarct on the radiogram because of the changes brought about by the pulmonary stasis simultaneously present.

As regards lung embolism without infarction VON DEHN has recorded a case he examined post mortem. According to this author the radiogram showed »besonders stark ausgeprägte Hilusschatten» without any sort of glands being found on autopsy in the hilum. About this form ASSMANN writes as follows: »Trombose oder Embolie der Lungengefässe ohne gleichzeitige Infarzierung des Lungengewebes dürfte zu einer Verstärkung der Schattenstrangzeichnung in dem betreffenden Bezirk führen. Ich selbst», writes ASSMANN, »habe keine derartigen Fälle beobachtet». Besides VON DEHN's case I have found nothing in the literature in reference to lung embolism without infarction.

Anatomy of the pulmonary vessels

Before describing the roentgen appearance of lung embolism I must give a brief account of the anatomy of the pulmonary vessels since this is decisive for the localisation and distribution of the changes.

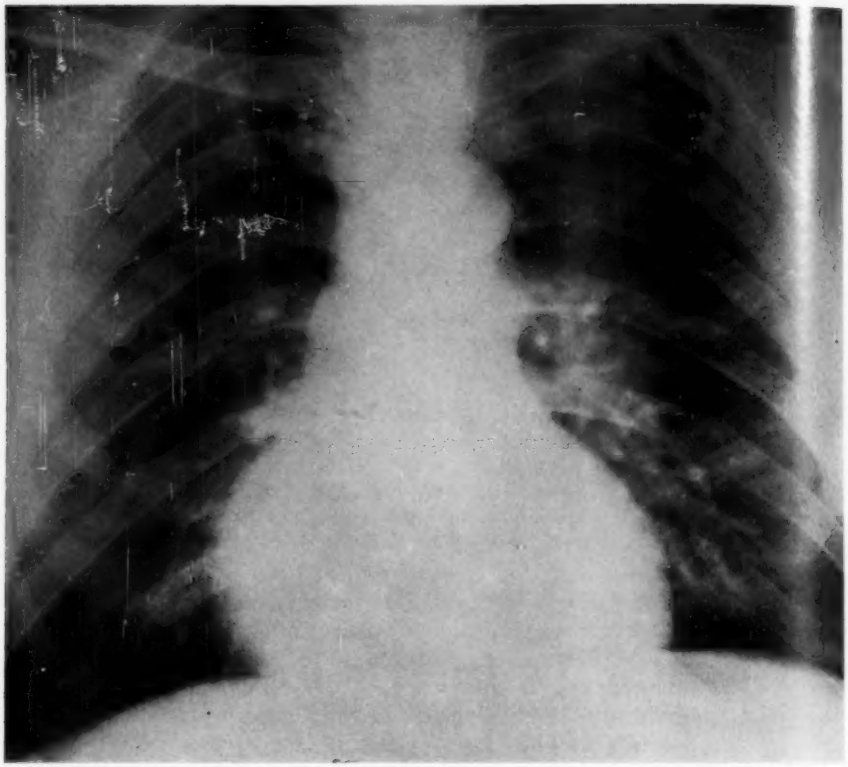


Fig. 4. Case 3. Examination after taken ill with embolism with stitches and sanguinolent expectoration. There is a decided clearing-up with inhibited vascularisation in the lower parts of the lower lobe. Centrally the vascular design appears normal but the vessels end suddenly when approaching the anaemic region.

A distinction is made between those branches of the pulmonary artery that have to do with the oxygenation of the blood and those of the bronchial artery concerned with the nutrition of the lung itself.

EWART, MELNIKOFF, FELIX, BACKMAN, HERNHEISER and KUBAT have investigated the distribution of the pulmonary vessels within the lungs. On the whole the arteries are found to follow the bronchi in their ramification into the lung. According to HERNHEISER that branch of the right pulmonary artery leading to the right upper lobe divides into apical, pectoral and axillary arteries. Of these the apical artery passes to the lung apex, the pectoral artery runs forwards and upwards towards

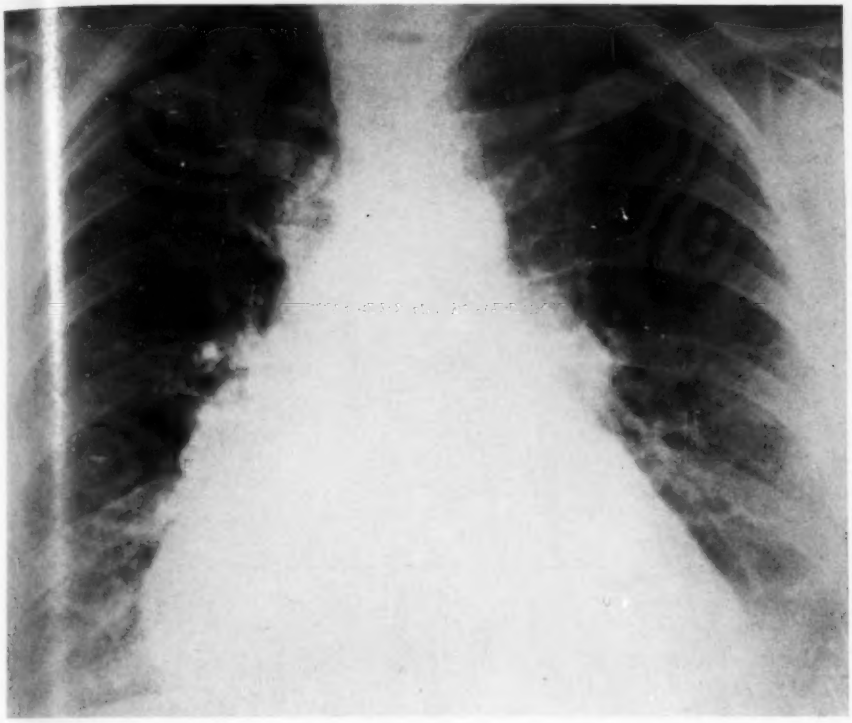


Fig. 5. Case 4. Examination before taken ill with pulmonary embolism. Well-marked cardiac hypertrophy with mild stasis of the lungs.

the anterior portion of the upper lobe and the axillary artery is seen to run backwards and outwards towards the posterior and lateral part of the upper lobe. The artery of the middle lobe divides into a medial and a lateral branch, ramus paramediastinalis and ramus costalis.

The artery of the lower lobe divides into an apico-horisontal branch running to the upper posterior part of the lower lobe and into antero-basal, postero-basal, anterior axillo-basal and posterior axillo-basal branches. These branches form ventral and dorsal pairs of arteries which run to above lying parts of the lung in a dorsal and ventral direction.

Thanks to this anatomical distribution of the pulmonary artery its ramifications will pass to regions mainly located as ventral or dorsal wedges in the lung. Changes arising through emboli will thus take the shape of wedges mainly assuming a dorsal or ventral direction.

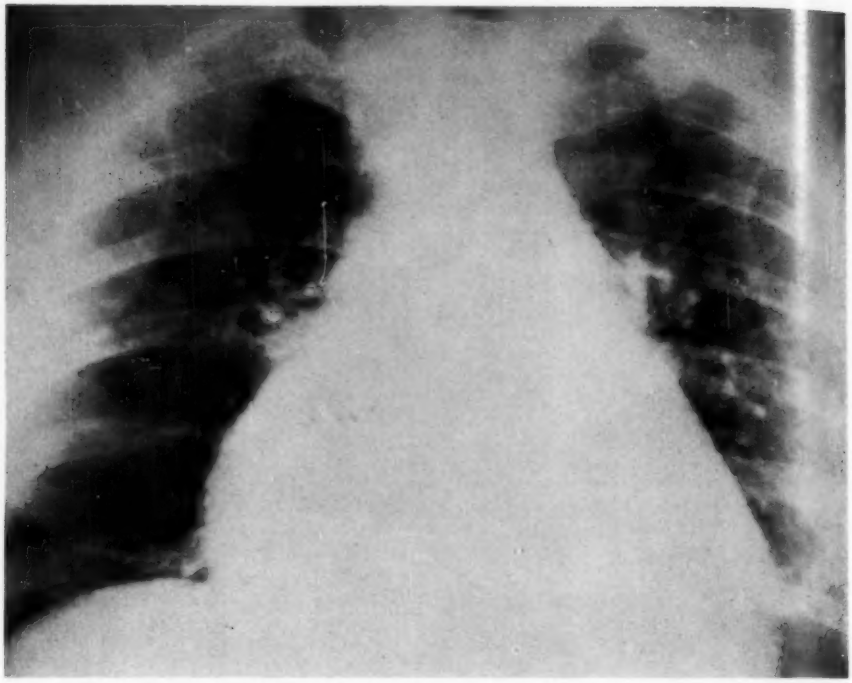


Fig. 6. Same case. Examination carried out immediately after onset of lung embolism with stitches, bloody sputum and dyspnoea. In the right lower lobe can be seen a wedge-shaped area with absence of vascularisation. The region is in evidence as a distinct clarification. Distinct stasis of left lung. Abrupt termination of vascularisation in right lung.

The author's observations of the roentgen appearance of embolism of the pulmonary artery

Material

Since 1933 I have had the opportunity of examining by roentgen 26 cases where embolism of the pulmonary artery had subsequently been found on autopsy. In all these cases the roentgen examination was carried out within a fortnight of the patient's death. Thus in 4 cases the examination was carried out on the same day that death occurred and in 8 cases within 2 days of death. In the majority of cases the examination was carried out on repeated occasions before death which enabled one to follow the development of the embolus. In these cases therefore one has

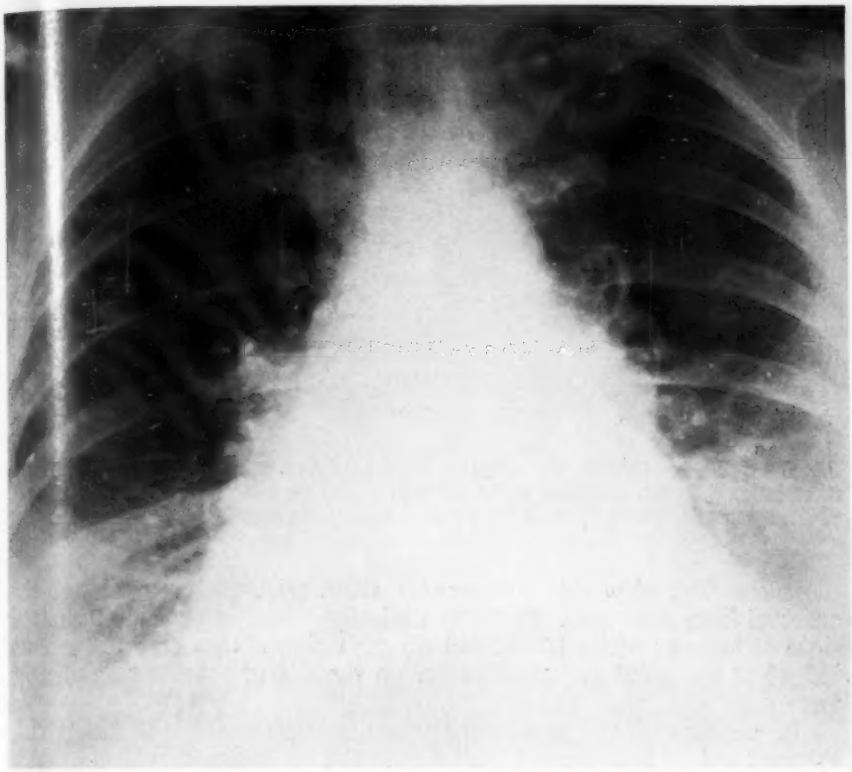
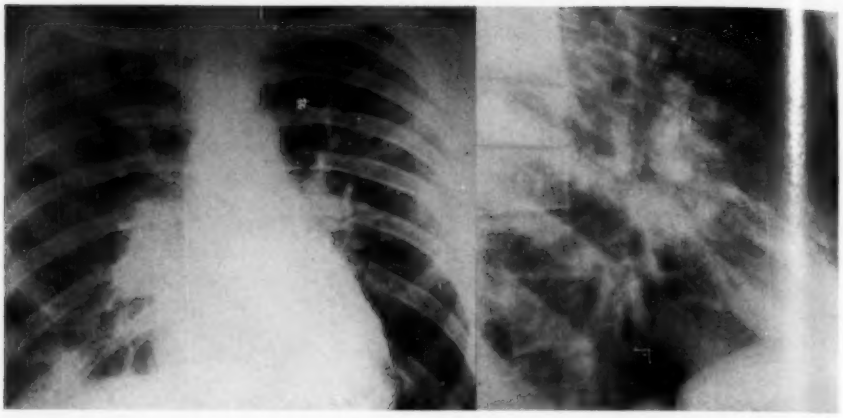


Fig. 7. Same case 3 weeks later. At the site of the previous clarification there is now a wedge-shaped massive shadow indicating the formation of infarct.

able to compare the roentgen findings with those of autopsy. Ten out of these 26 cases showed signs of lung infarction while the rest presented signs of more or less widespread embolism without infarction. Regarding the 10 cases with infarcts 7 of them showed signs of lung embolism involving larger portions of the lung than represented by the infarcts observed. Cases presenting complete obstructive lung embolism have shown either no signs of infarcts at all or only such of minor degree. My study of the progress and possible healing of emboli has also been based on 18 further cases with a clinical picture of lung embolism where the roentgen appearance was similar to that present in the autopsic cases. In these cases one was able to compare the radiograms during the embolic stage with pictures obtained during the process of healing and after recovery. In some



a. b.

Fig. 8 a and 8 b. Case 5. Examination immediately after onset of embolism. In the right lower lobe can be seen a wedge-shaped massive shadow. In the left lung there is obvious anaemia, particularly at the base. Centrally the vascular design ends abruptly.

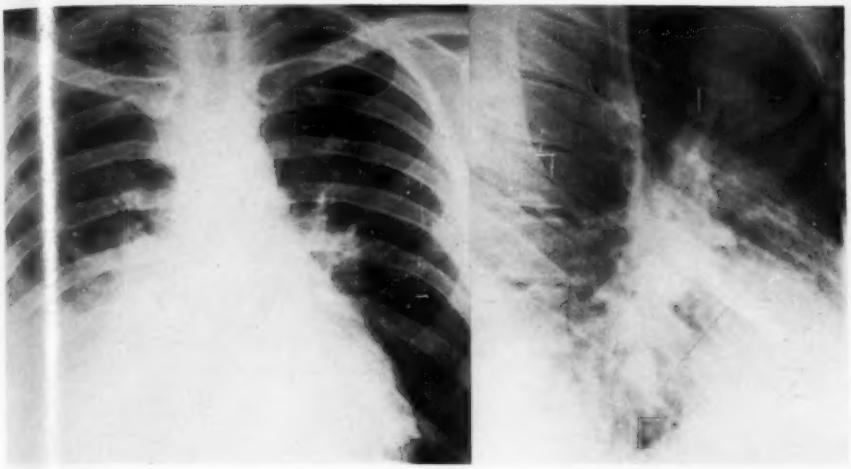
cases one was also able to compare them with pictures prior to the onset of lung embolism. Of these clinically observed patients 7 showed signs of infarcts while 10 showed no such signs. One case showed no infarct at the roentgen examination on the day of onset but a fortnight later such signs were present.

In embolism of the pulmonary artery we are thus able to distinguish between emboli without and emboli with the formation of infarcts. Each one of these two forms has got its own characteristic roentgen appearance.

The roentgen appearance in embolism of the pulmonary artery without infarction

In uncomplicated cases of embolism of the pulmonary artery and its branches we get, as above mentioned, ischaemia of the lung area corresponding to the embolised arterial branch. The branches of the pulmonary artery are devoid of blood and contracted on the peripheral side of the embolus. The lung region however receives nourishment from branches of the bronchial artery so that no necrosis needs arise. On the other hand there is probably no exchange of gases within the embolised lung region.

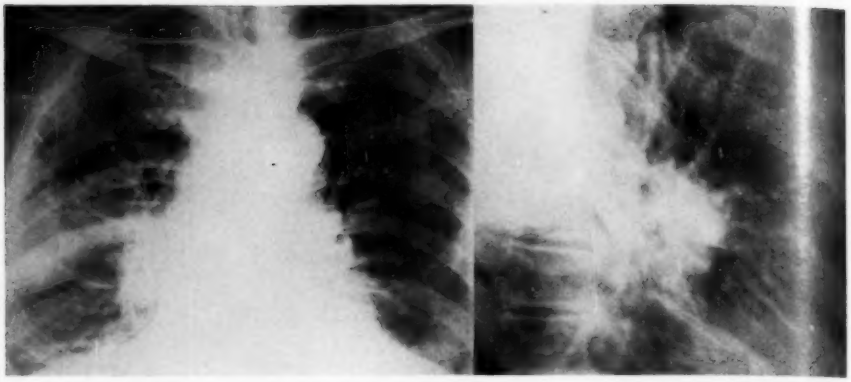
In those cases showing lung embolism without infarction the ischaemia of the distributed area of the pulmonary artery on the peripheral side



a. b.

Fig. 9. Same case 10 days later and the same day as death occurred. The infarct shadow in right lower lobe has become somewhat larger. In addition right-sided pleurisy. In the whole of the left lung and also in parts of the right upper lobe there is well-marked anaemia with diminished vascular design. The central vessels are seen to end abruptly in the anaemic area.

of the embolus or emboli has appeared on the radiogram as a local and well defined clarified zone with diminished or absent vascularisation. In an area corresponding to the site of the embolus and on the central side of this the vascular design has been well maintained but seems to stop abruptly. On account of the distribution of the arterial branches the clarified zone assumes the shape of a wedge with its apex directed towards the hilum and its base towards the periphery of the lung. The axis of the wedge generally passes obliquely forwards or obliquely backwards. With suitable projection the shape of the wedge most often appears on lateral or semi-lateral pictures. Lesser wedges may also appear on antero-posterior pictures. In the case of larger emboli a whole lobe or an entire lung may be anaemic. In these cases it is only the vascular design in the central parts of the lung that can be distinguished while the peripheral area is anaemic with inhibited or diminished vascularisation. There are a few finer strands to be seen there probably corresponding to the branches of the bronchial artery. This clarified area in the lung design is more clearly in evidence in those cases where a congested lung is simultaneously present. It is clear, therefore, that lung embolism without infarction leads to anaemia and diminished vascularisation and not hyperaemia and



a. b.

Fig. 10 a and 10 b. Case 6. Examination carried out after onset of lung embolism 2 days before death. A wedge-shaped masisve shadow can be distinguished in the upper posterior part of the right lower lobe. In right upper lobe and the left lung there is obvious anaemia with diminished vascular design. The central vessels end abruptly.

increased vascularisation as assumed by previous authors, such as Assmann and Frimann-Dahl.

An embolus not leeding to infarction may become organised and absorbed whereby the circulation in the pulmonary artery is reestablished. The vascular design then returns to the formerly anaemic lung region and the transparent zone disappears.

On the other hand thrombotic masses may become deposited on a smaller embolus causing this to enlarge in a central direction. This causes still larger parts of the lung to become anaemic. Such a progress of the embolus may lead to complete occlusion of the pulmonary artery with subsequent fatal issue. A continuous increase of the anaemic area of the lung is generally indicative of a poor prognosis.

Regarding the differential diagnosis one has in the first place to take into account bronchostenosis with valvular action. In this condition we also get wedge-shaped areas in the lung with a definite clearing-up as a result of emphysema and anaemia. In well marked cases of that nature the wedges however have a clearly convex outline and can thus be distinguished from lung embolism. In cases of mild degree however great difficulties may hamper a correct diagnosis. The presence of a central vascular design which ends abruptly seems to be favour of embolism. In difficult cases it is advisable to do a bronchography by which the presence or absence of bronchostenosis can be ascertained.

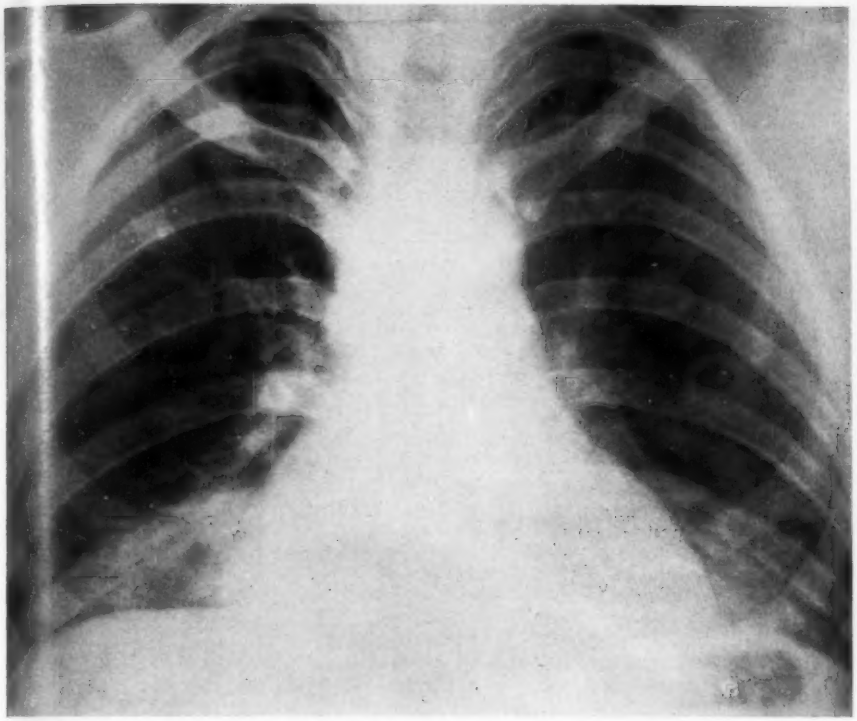


Fig. 11. Case 7. Examination carried out after onset of lung embolism. In the basal parts of both lower lobes there are wedge-shaped massive shadows.

The roentgen appearance in embolism of the pulmonary artery vith infarction

The lung infarct appears on the radiogram in a suitable projection as a wedge-shaped massive homogenous shadow. The wedge has its apex directed towards the hilum and its basis towards the periphery of the lung. In all my cases the infarcts have been located in the posterior parts of the lung causing the axis of the wedge to be directed backwards. Yet the infarct can also be directed forwards and be located in the anterior parts of the lung. In infarcts of smaller size the axis of the wedge may also run in a more frontal direction causing the shape of the wedge to be observed even on an antero-posterior picture. Larger infarcts present their wedge-shape best on a lateral picture or else on a picture with oblique direction of the rays preferably adjusted under screening. The wedges are

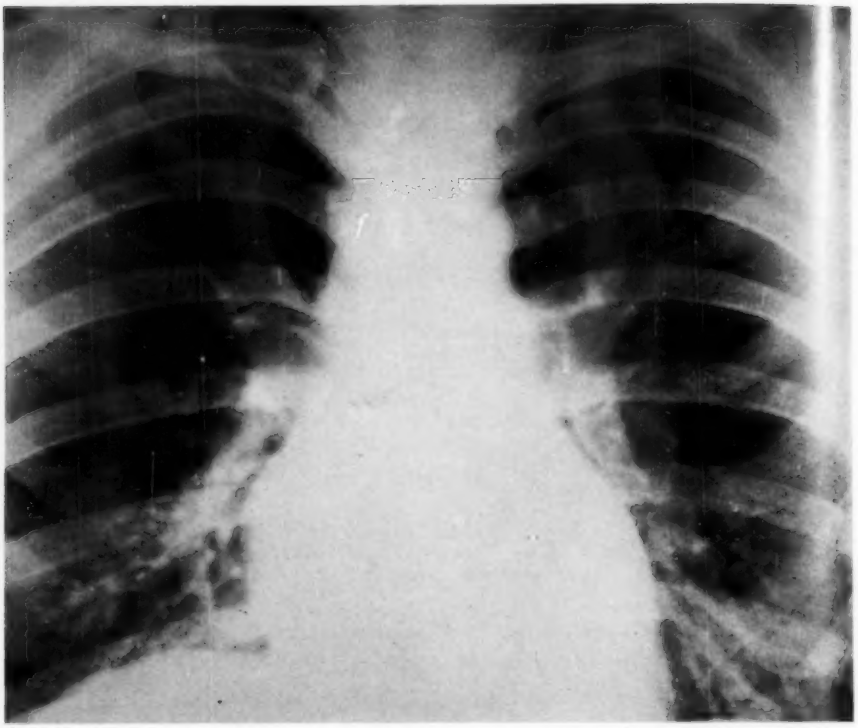


Fig. 12. Same case a fortnight later. The wedge-shaped shadows have completely disappeared.

generally sharply defined and present a clear and smooth bordering surface.

Since the infarct exclusively seems to occur in connection with stasis of the pulmonary circulation we generally get a more or less congested lung simultaneously with the infarct. The changes produced by the stasis may wholly or in part cover the infarct shadow so as to render the wedge-shaped shadow indistinguishable. Provided there is no pronounced stasis with pleural effusion it is generally possible to distinguish, by using a suitable projection, the wedge-shaped shadow produced by the infarct.

The infarct may become completely absorbed provided the infarcted lung area be not too large and has not been subject to complete necrosis but nourished by branches of the bronchial artery or by newly formed

vessels. During this stage of absorption the infarct more and more resumes its aerated condition. It then no longer appears on the radiogram as a homogeneous wedge-shaped shadow but assumes a reticular appearance.

What usually happens however is that the infarct does not get absorbed but dies off and undergoes organisation. This gives rise to a reactive inflammatory zone surrounding the infarct. The wedge-shaped character then disappears and the shadow assumes a more diffuse outline. Gradually the infarcted area undergoes cicatricial transformation, wedge-shaped or linear band-shaped changes arising. On the radiogram these changes appear as band-shaped or narrow wedge-shaped shadows in the lung radiating from the hilum to the periphery of the lung.

Besides the infarct 7 out of the 10 infarctic cases have shown signs of larger or smaller anaemic areas with the vascular design altogether absent or diminished in the peripheral part of the lung and with retained but abruptly terminating vascular design on the central side of these areas. In one and the same case therefore we may often find one embolus with infarction and another embolus without infarction. The explanation of this will be subject to subsequent investigations.

Regarding the differential diagnosis we have in the first place to consider bronchial occlusion with a secondary obstructive atelectasis. This too gives rise to a wedge-shaped homogeneous shadow of similar localisation and appearance as the infarct. In bronchostenosis, however, at any rate in its early stages, the contours of the atelectatic infil ration are concave while the infarct presents a more even outline. In complicated infarcts the differential diagnosis may be difficult also in regard to bronchopneumonia and bronchitis. By careful examination in different projections it is possible, however, to recognise the typical wedge-shape of the infarct. Should there be any difficulty in distinguishing between infarct and bronchostenosis it is advisable to do a bronchography. Such examination enables us to verify or exclude the presence of bronchostenosis. As mentioned above signs of lung embolism without infarction are often also present in these cases of lung embolism with infarction whereby it will be possible to make a diagnosis.

To diagnose roentgenologically embolism of the pulmonary artery is very difficult, particularly in cases without infarction. The greatest difficulty in these cases is really to decide whether some change is present or not. Even in pictures of normal lungs we sometimes get a sudden interruption of the vascular design owing to the fact that the vessels on division take another direction. On careful analysis of the radiograms taken in different directions and preferably on repeated occasions it is generally possible however to arrive at a correct diagnosis. Where older radiograms

are in existence a comparative study of these may be helpful. In the case of older patients suffering from some chronic disease, particularly heart disease, one should always bear in mind the possibility of embolism of the pulmonary artery.

SUMMARY

In embolism of the pulmonary artery a distinction can be made between emboli without and emboli with infarction. Each one of these forms presents its own typical roentgen appearance.

In embolism of the pulmonary artery without infarction we get ischaemia of the branches of the pulmonary artery on the peripheral side of the embolus. On the radiogram this ischaemia appears as a clarified area with diminished vascular design corresponding to the extent of the embolised artery. The vascularisation is however maintained in the central parts of the lung. The vascularisation takes however a rapid end to pass over into the above mentioned area of non-vascularisation.

The infarct appears on suitable projection as a wedge-shaped massive homogeneous shadow. In most cases, however, where infarction is present one is able to observe also within other parts of the lung larger or smaller wedge-shaped clarifications as in embolism without infarction.

ZUSAMMENFASSUNG

Bei Embolie der Lungenarterie kann zwischen Embolie ohne oder mit Infarktbildung unterschieden werden. Jede von diesen Formen bietet ihre eigene typische röntgenologische Erscheinung.

Die Embolie der Lungenarterie ohne Infarkt ergibt sich Ischämie der peripher vom Embolus gelegenen Pulmonariszweige. Auf dem Radiogramm erscheint diese Ischämie als ein aufgehelltes Gebiet mit verringerter Gefässzeichnung entsprechend dem Gebiet der durch den Embolus verschlossenen Arterie. In den zentralen Teilen der Lunge ist die Vaskularisation erhaltengeblieben. Beim Übergang in das obenerwähnte nichtvaskularisierte Gebiet nimmt die Vaskularisation jedoch ein rasches Ende.

Der Infarkt erscheint bei geeigneter Projektion als ein keilförmiger massiver homogener Schatten. In den meisten Fällen, wo ein Infarkt vorliegt kann man jedoch auch an anderen Teilen der Lunge grössere oder kleinere keilförmige Aufhellungen sehen, wie bei Embolie ohne Infarktbildung.

RÉSUMÉ

Une distinction peut être faite, parmi les embolies dans l'artère pulmonaire, entre l'embolie avec infarctus et l'embolie sans infarctus. Chacune de ces formes présente un propre aspect radiographique typique.

L'embolie sans infarctus nous donne une ischémie des branches de l'artère pulmonaire sur le côté périphérique de l'embolie. Cette ischémie apparaît, au radiogramme, comme une surface éclaircie, avec dessin vasculaire diminué correspondant à l'étendue de l'artère embolisée. La vascularisation subsiste cependant dans les parties centrales du poumon. Elle cesse toutefois rapidement et passe en la surface de non-vascularisation précitée.

L'infarctus apparaît, par projection convenable, comme une ombre cunéiforme massive et homogène. Dans la plupart des cas avec infarctus, cependant, il est possible d'observer également en d'autres parties du poumon des éclaircies cunéiformes plus ou moins grandes, comme dans l'embolie sans infarctus.

FROM THE ROENTGEN CLINIC OF THE KOMMUNEHOSPITAL, COPENHAGEN
(CHIEF: SENIOR PHYSICIAN, DR. GEORG BIERING)

SOME CRANIAL CHANGES IN RECKLINGHAUSEN'S NEUROFIBROMATOSIS[1]

by

Thomas Rosendal

The bone changes in Recklinghausen's neurofibromatosis were discussed in an article, by NØRGAARD, in the May number of the Acta Radiologica for 1937, illustrated by two cases: one with pronounced kyphoscoliosis, the other with bone cysts in the femur. In the same paper a detailed description was given of the disease, whose typical symptom is the cutaneous neurofibromas or pigmentations. I shall therefore here only enumerate briefly the osseous changes mentioned in the literature as apt to occur; referring, for the rest, to SCHINZ-BAENSCH-FIEDL's (1932) and NELSON's (1936) text-books of radiology, and to KIENBOECK and ROESSLER's monograph, in which are collected the numerous reports, up to 1932, of such changes. They may consist in congenital malformations, bone defects or partial atrophy of bones, specially localised to the skull and extremities; more rarely in hypertrophy or anomalies of growth. Finally, there may be pronounced kyphoscoliosis, probably on the basis of osteoporosis; or isolated wear on some bone or other, resulting from pressure of a tumor, sometimes manifesting itself as a subperiosteal cyst, specially in the long tubular bones.

The present communication deals with the cranial changes, of which there are but few reports in the literature. KIENBOECK, for instance, in the monograph just referred to, has only been able to collect about 15 such cases examined by roentgen. Therefore, the fact that it has been possible to collect no fewer than 8 more, which during the period 1934—38 have been admitted to Copenhagen hospitals with symptoms of neurofibromatosis, and in all of which cranial changes illustrating various types have been found, seems to be worthy of attention, and to justify their

[1] Read in a somewhat shorter form at the meeting of the Danish Radiological Society April 7th, 1938. — Submitted for publication July 6th, 1938.

presentation here. Besides, it must be supposed that the cranial changes, precisely in neurofibromatosis, are of interest from the viewpoint of roentgen diagnosis, because in that disease, according to BUMKE and FOERSTER (1936), intracranial processes may be found, such as neuroma of the acusticus, specially with bilateral localisation; glioma of the optic nerve, tumors of the brain stem and cerebrum, multiple meningeoma, and, finally, diffuse arachnoiditis.

For the sake of better survey, I shall speak of the eight cases in the following order. First 2 which show cranial changes localised to both optic foramina, then 1 with abnormal vascular design in the skull and a curious depression in the sella turcica, and lastly 5 with extensive atrophic changes of the skull.

The two first have already been the subject of communications from Drs. BUSCH and CHRISTENSEN, of the neurosurgical clinic of the State Hospital in Copenhagen, to the Zentralblatt f. Neurochirurgie (nos. 5—6, 1937). I thank Dr. BUSCH for permission to speak of them here. Both patients were girls, 7 years old. In one case, the mother and the maternal grandmother had typical neurofibromatosis; in the other there was no information of that disease in any other member of the family. Both patients had light coffee-colored pigmentations of the trunk and extremities, as indications of an identical affection. In one of them, the vision on both eyes became gradually reduced in the course of six months; in the other, the same symptom developed in the course of three years. In both cases, neurological examination showed bilateral atrophy of the optic nerve, but no other neurological symptoms. In both cases also, roentgen examination showed a bilateral widening of the optic foramen to three times the normal size. In both cases, cylindrical tumors were, on craniotomy, found in the optic nerves, together corresponding to the optic chiasm. Large portions of these tumors were removed, with resulting improvement of the vision. Histologically, the growths were composed of a cell-type, oligodentrocytes, which may be formed by differentiation from the glia-cells of the optic nerve. The oligodendrocytoma thus belongs to the group of gliomas. I quote these cases as illustrations of the isolated cranial change produced by the pressure from a tumor, and at the same time as examples of the occurrence of a special brain tumor in connexion with Recklinghausen's neurofibromatosis.

The next case represents another type of neurofibromatosis. The patient, a man aged 40, had since childhood suffered from numerous skin tumors. Biopsy on one of these, in 1938, showed it to be a neurofibroma. Also the cranial changes in this case are different from those just described; what they correspond to, pathoanatomically, cannot be determined with certainty. I bring the case forward in order to show how the pathological picture of neurofibromatosis can vary, and also t

illustrate the cranial changes in a case of supposedly diffuse meningeal affection. The man has since his eighteenth year had a stationary kyphoscoliosis, developed in the course of six months following a lesion to this back. Besides, he has for two—three years had some indefinite trouble with his right knee. Roentgen examination has shown a thickening of the right femur, just above the condyles, with a depression on the posterior side and, about the latter, signs of periosteal neoformation of bone. Finally, he has a circumscribed indolent, walnut-sized tumor, judged to be a neurofibroma, in the right side of the neck. These three affections can be naturally explained on the basis of neurofibromatosis; the two first are examples of the bone changes in the spine and extremities which, as already said, may be found in that disease.

Fig. 1.

Since December, 1937, the man has been a patient first in the Kommunehospitalet, afterwards in the neurosurgical clinic of the State Hospital. He was admitted originally owing to repeated attacks of vertigo, from which he had been suffering for two years back; during the last months there has been added to these a constant diffuse, pressing cephalgia, most pronounced in the frontal region. Repeated special examinations have failed to disclose any signs of brain tumor, particularly of hypophyseal tumor. Nevertheless, the roentgen examination of the skull shows changes which might point to the latter. In the lateral view (Fig. 1), we see the large, markedly deep sella turcica, the bottom of which seems to be in some way depressed. It doesn't quite look like a »pressure sella»; nor does it show any such changes as we shall find in the following cases. The anterior clinoid processes are short and plump, the dorsum sellæ

steep, but not curved backwards; the osseous structure is not atrophied. The sphenoparietal sinuses are very broad, the left perhaps a little larger than the right. The vascular design corresponding to the left parietal bone is fairly well pronounced. Ventriculography has shown the whole system to be slightly distended, with the third ventricle normal; a suprasellar tumor is therefore out of the question. Incidentally to the same investigation, the cerebral membranes corresponding to the points of puncture were found to be thicker than normal.

Fig. 2.

The result of the roentgen examination suggests the possible presence of small diffuse meningeomas corresponding to the cranial convexity. This, as we have said, is a characteristic form of intracranial tumor in neurofibromatosis. Or the findings may denote a diffuse alteration of the membranes of the brain; another condition which is sometimes found in these cases, and which manifests itself by what is called arachnoiditis; that is, a thickening of the membranes with ingrowth of vessels into the normally avascular arachnoidea. That such a thickening was found, incidentally to the ventriculography, at the points of puncture for the latter, would seem to speak for the last named diagnosis as the correct one. For the enlargement of the sella I cannot find any adequate explanation; possibly the abnormal vascular conditions may have something to do with the matter.

The next five cases are all characterised by atrophic cranial changes. Before discussing the latter, I briefly record the case history of each.

No. 1. — Boy, 14 years old; no other case of neurofibromatosis traced in the family. — The patient has always had light coffee-colored pigmentations on the trunk and extremities. Since early childhood, his face has been marked by the abnormal features seen in Fig. 2; namely a soft intumescence laterally in the left upper eyelid and a slight elephantiac hypertrophy of the left side, extending backwards and dislocating the left ear downwards. In the left buccal mucosa there is a hazelnut-sized, soft intumescence; the left side of the pharynx is asymmetrical, with some outbulging of the tonsillar bed and pharyngeal arches. Behind the left ear, the mastoid process is felt to be atrophic, and the bone posterior to it likewise irregularly atrophic, with several depressions. In 1938, he seeks the ophthalmological policlinic of the Kommunehospitalet on account of his eyelid. There, the intumescence of the latter is judged to be a neurofibroma. Neurological examination discloses paralysis of the left glossopharyngeus nerve, but otherwise nothing that might point to the presence of an intercranial tumor. On the other hand, he is almost completely deaf on the left ear, and there are secondary changes there after an ot[illegible]s

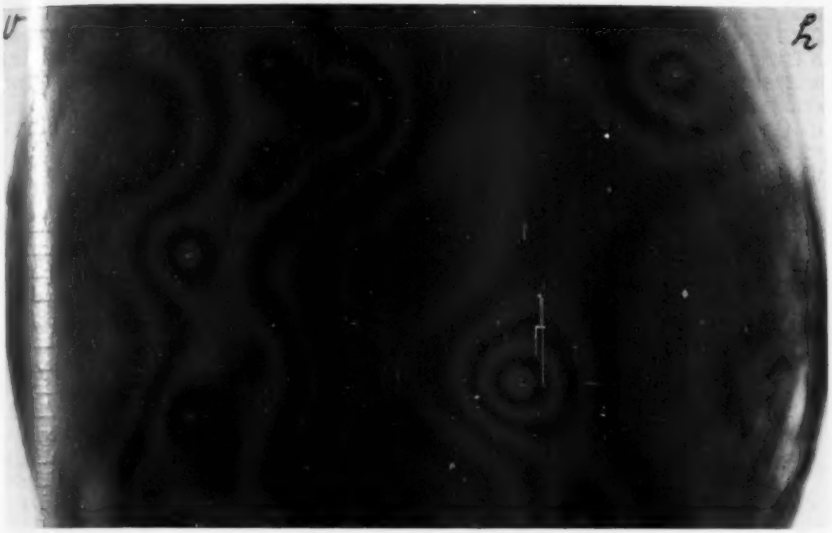


Fig. 3.

media. Roentgen examination of the cranium shows in occipital projection (Fig. 3) very considerable atrophy, localised to the posterior portion of the left temporal bone, corresponding to the region which is felt atrophic under the skin. There is also considerable atrophy of the left mastoid process, where the pneumatisation is to a great extent obstructed; and the whole of the pars petrosa is atrophic, as well. In a roentgenograph of that region alone, it is seen that the acoustic pore is neither dilated nor eroded. In the asymmetrical foramen magnum is seen a pronounced spina bifida corresponding to the posterior arch of the atlas. In the axial projection (Fig. 4) is seen the atrophy, just mentioned, of the left pars petrosa, whose

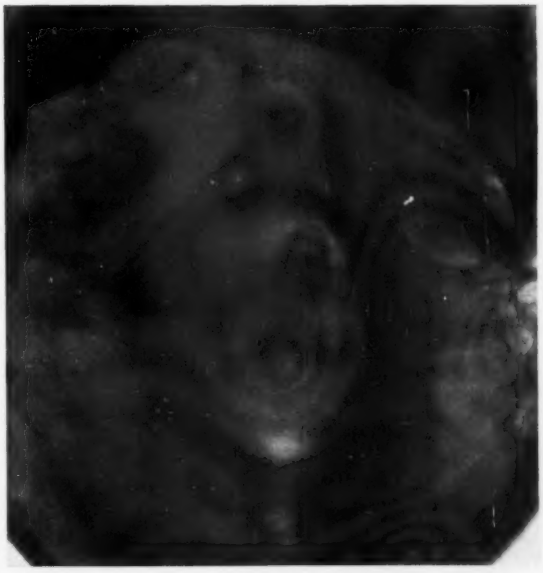


Fig. 4.

osseous structure and contour stand out distinctly, surrounded by a bone-free margin from 0.5 to 1 cm. broad, corresponding to the foramen lacerum. We furthermore see a fairly pronounced diastasis between the anterior left contour of the occipital bone and the posterior margin of the left ala magna; which makes the foramen lacerum appear enlarged. In this projection there is, besides, a marked asymmetry of the left half of the mandible, the ramus of which is shortened and dislocated anteriorly. Finally, there is some atrophy of the facial bones of the left side; but no enlargement either of the orbits, the left middle cranial fossa or the sella.

Fig. 5.

No. 2. — Girl, 17 years old. The father is stated to have had scattered soft tumors on various parts of the trunk. — The patient herself has numerous light coffee-colored pigmentations on the trunk and extremities. Since her birth, she has had a diffuse, elephantiac hypertrophy of the right side of the face. She has been repeatedly operated on for this, and now has several cicatrices on her cheek. As seen in Fig. 5, there is considerable hypertrophy of the right half of the face, with deformation of the eye-region, mouth and nose. In addition to this, there is hypertrophy of the gingiva of the right half of the upper jaw. In 1938, she consults the ophthalmological clinic of the State Hospital, as the deformation of her right eyelid interferes with the vision. On examination, both eyes are found to be normal. A neurological investigation showed nothing indicative of intracranial affection. Roentgen examination of the cranium shows in frontal projection (Fig. 6) asymmetry of the right half of the face, and narrowing of the right orbit, with atrophy of the lateral margin. The foramen rotundum of the right side cannot be demonstrated. The cranial wall of the temporal region is thinner than normal. The right half of the mandible is narrowed and attenuated. In axial projection there is noticed, besides, an effacement of the normal structure posteriorly in the right ala magna, where neither the foramen ovale nor the foramen spinosum can be seen. The petrous parts of two temporal bones are alike. The region of the sella is normal. The design of the sutures and the digitate impressions is not abnormally pronounced.

No. 3. — Man, 38 years old. Can give no information pointing to other cases of neurofibromatosis in the family. — The patient has always had light coffee-coloured pigmentations on the trunk and extremities. Since early childhood also in the left temporal region a large, soft intumescence, which has grown steadily, also after it was extirpated in 1928. In 1938, it was extirpated again, in the Kommunehospitalet, and on that occasion there were found, corresponding to the swelling, numerous cord-like formations, as thick as slate-pencils, just as in the case of a plexiform neurinoma. Microscopy showed a benign mesodermal tumor, partly with connexion to nerve elements. After the operation, the facial deformation is as seen in Fig. 7. Eye- and neurological examinations have shown normal conditions. Otological examination showed reduced

Fig. 6.

Fig. 7.

Fig. 8.

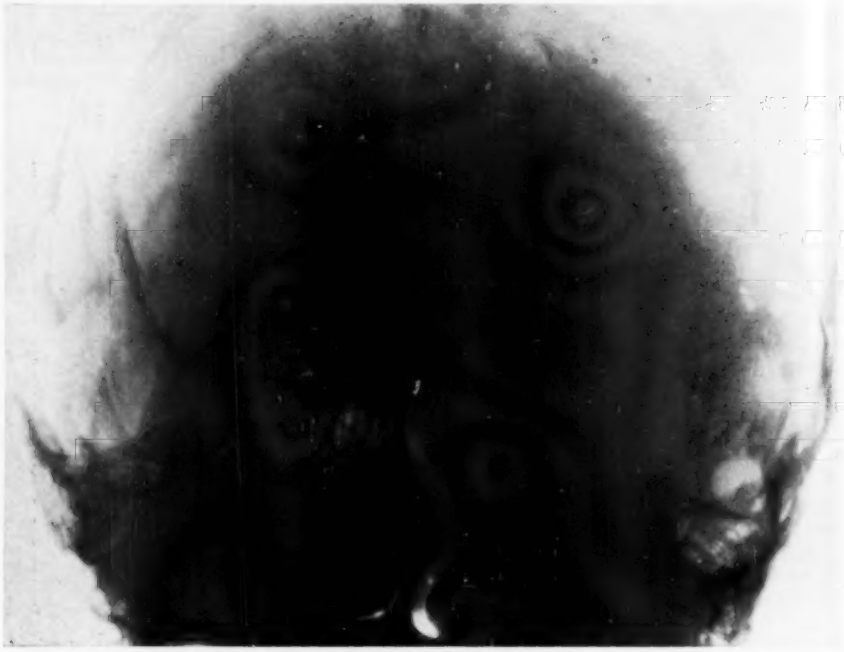


Fig. 9.

hearing on the left side, which can be explained by the dislocation downwards of the external ear, seen in Fig. 7. Roentgen examination of the skull shows asymmetry of the left orbit, which is narrower and somewhat more elongated than the right, with atrophy of the lateral margin and the crista temporalis. The cranial wall of the left temporal region, backwards to behind the ear, is thinned down unevenly, and in axial projection it is seen that the atrophic process is localised to the external side of the cranial bones. Also the left mastoid process is atrophic. There is no enlargement of the middle cranial fossa, the orbit or the sella turcica. (For illustration see Fig. 6 of the preceding case, the cranial changes in which are of nearly the same type, localised to the upper right temporal region).

No. 4. — Boy, 17 years old. No familial record of neurofibromatosis.[1] — As Fig. 8 shows, there is congenital hypertrophy of the left upper eyelid. On palpation, there can be felt vermicular cords, and something like a pedicle extending into the orbit, laterally, beneath the roof of the latter. Besides, there is some hypertrophy of the left half of the face, with pigmentation; and a flat protuberance, the size of a child's hand, in the left temporal region. On the trunk, there are scattered, light coffee-colored pigmentations. In 1927, and again in 1936, he was operated on, in the ophthalmological department of the State Hospital, for the hypertrophy of the eyelid, and nodular formations were found.

[1] Journal and films kindly lent by the ophthalmological dep't of the State Hospital.

Microscopy of the latter showed the mass to consist of connective tissue, possibly with an immixture of nerve tissue (neurofibroma).

Already before this, in 1922, when he was about 1 year old, the boy had been operated upon in the State Hospital (dep't D) for the above named intumescence in his left temporal region, which, as it seems, had been developing strongly during the time immediately preceding, and which was taken as an indication of cerebral tumor. When he was admitted, it was about the size of a watch-crystal, with a pulsating defect, about 1×2 cm. large, corresponding to the fontanel. On trepanation, the intumescence was found to correspond to an outward bulge of the anterolateral fontanel, which was surrounded by thin, soft bone edges. When the dura mater was pierced, there came a considerable outflow of cerebrospinal fluid, and a dull-white, jelly-like, half-transparent mass was seen, from the surface of which drops oozed forth. After compression, the surface of the brain became visible, and a cavity between the latter and the cranium. No cerebral tumor was found, and no diagnosis was made. The bulge referred to has not become larger since.

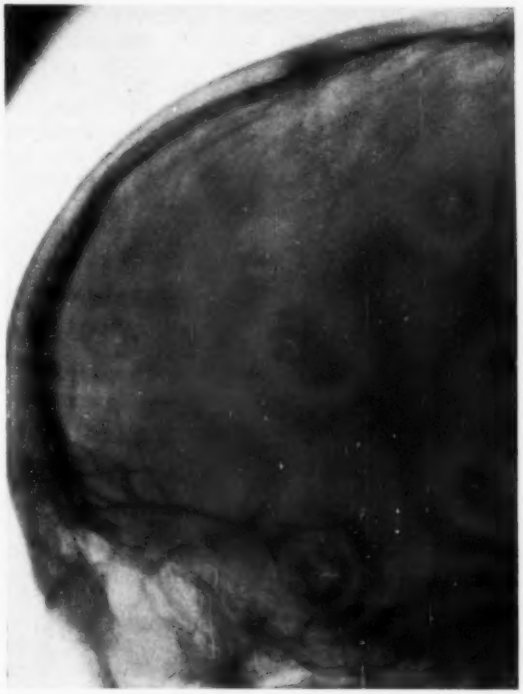


Fig. 10.

On examination during the patient's two stays in the State Hospital, in 1927 and 1936, as well as on ambulant examination in the Kommunehospitalet in 1938, the left eyeball was found to be normal, but dislocated downwards, with distinct pulsation. There was convergent strabismus, and the vision on the left eye was reduced. Ophthalmoscopy showed nothing abnormal, specially not in the way of papillary changes; the strabismus would account for the reduced vision on the left eye. Repeated neurological examinations have failed to reveal any indications of intracranial tumor; there are no signs of acromegaly, nor any dysendocrine symptoms.

The pathological features of the cranium seen in the roentgenographs from 1927, 1936 and 1938 are identical, and may thus be considered as stationary. In the frontal view (Fig. 9) the left orbit is seen to be enlarged to nearly double its natural size, with displacement of the margins in all four directions. The left half of the sphenoid bone is partly lacking, partly so thin that its osseous structure is almost effaced. Neither the superior or inferior orbital fissure nor the left ala parva can be distinguished. The superior border of the left ala magna, which forms the boundary between the anterior and

middle cranial fossæ, is equally indistinguishable, and so is the left foramen rotund m. Through the dilated orbit is seen the atrophic pars petrosa. Corresponding to the eft temporal region there is a diffuse prominence, as large as a palm, of the cranial v ll, which is markedly and unevenly thinned down corresponding to the interior side. he atrophy extends backwards into the parietal bone, and even in the temporal bone t ere are a few irregular rarefactions. In the lateral view (Fig. 10), the sella turcica is see to be enlarged to at least four times its natural size. Specially the enlargement of the eft side is great, and the left anterior clinoid process is not seen. The sphenoidal s us cannot be made out. The bone contour which m rks the partition between the left orbit and the mi dle cranial fossa is just barely visible, and seems to be displaced forwards and downwards. Finally, there is in one axial view seen a sharp-edged defectouosity corresponding to the left ala magna, with effacement of the left foramen ovale. The design of the sutures and digitate impressions is not abnormally prominent.

Fig. 11.

No. 5. — Boy, 14 years old[1]. The mother and maternal grandmother stated to have suffered from soft tumors on the extremities. — The patient was admitted to the neurological department of the State Hospital in 1932. The cranial changes observed at that time were more pronounced even, than those in the preceding case. As Fig. 11 shows, there was an enormous elephantiac, congenital deformation of the entire right side of the face. Also the gingiva of the right upper and lower jaw was hypertrophied. The skin on the right side of the face was pigmented, and there were scattered, light coffee-coloured pigmentations on the trunk. The right eyeball was dislocated downwards and forwards, and the vision on the eye much reduced. The rest of the examination showed nothing abnormal; no signs of brain tumor; no dysendocrine symptoms. In 1934, the right eyeball was extirpated in the »Cripples' Home». The journal states that the roof of the orbit was paper-thin, but says nothing about the bulbus, or about tumors in the orbit. The patient died shortly after the operation. There was no post-mortem.

Roentgen examination of the skull shows pathological changes similar to those in the preceding case, only on the opposite side. The roentgenographs of that case may therefore serve as illustrations here, as well. In frontal aspect, the right orbit is twice as large as the left, all four margins displaced, the superior and lateral blurred. The normal design of the posterior wall is effaced, the bone contours of the right ala parva and ala magna being invisible, while the corresponding contours on the left stand out in normal design. The cranial wall above and laterally from the right orbit, corresponding to the right temporal region, is thin, with vaguely defined rarefactions; but there is no bulge, as in the preceding case, corresponding to that region. Finally, there is diffuse atrophy of the facial bones of the right side, and also the right half of the lower jaw is atrophic. The lateral view shows, like in the preceding case, a very considerable asymmetrical enlargement of the sella turcica, specially of the right side. The dorsum sellæ is bent slightly backwards, the sphenoidal sinus cannot be distinguished. Finally, the bone contour which marks off the middle cranial fossa anteriorly is partly effaced, and is displaced fo-

[1] Journal and films kindly lent by the neurological dep't of the State Hospital.

war[illegible]s on the right side. The cranial sutures are not widened; the digitate impressions not abn[illegible]rmally prominent.

Before discussing the five cases here described, it may be of interest bri[illegible]ly to consider the opinions expressed in the literature concerning the cra[illegible]ial changes in Recklinghausen's neurofibromatosis. The under-sta[illegible]ling of their pathogenesis has been difficult owing to the scarcity of r[illegible]entgen-examined cases and the equal rarity of published post-mortem rep[illegible]rts.

In 1924, VOGT reported 3 cases of unilateral elephantiasis of the eye[illegible]d, all with enlargement of the sella turcica. In his opinion, the enla[illegible]gement of the sella would be the manifestation of some hypophyseal affe[illegible]tion, while the facial hypertrophy represented the isolated acro-meg[illegible]aly. This report suggested to WINKELBAUER the publication, in 192[illegible], of a critical review of 9 cases collected from the literature of 1906—192[illegible], including the three just mentioned of VOGT's. All were cases of Recklinghausen's disease with pigmentations or skin tumors, and in them all there was unilateral elephantiasis of the face or eyelid. In some there were palpable nodules or string-like formations as signs of plexiform neuroma. From the published roentgen examinations, which are not all equally detailed, it appears that in 7 cases there was enlargement of the sella (whether asymmetrical or not is not stated), while in 1 case the sella had diminished in size. In 2 cases there were, besides, cranial hypertrophy, and in all the others cranial atrophy, on the same side as the elephantiasis. Finally, it is noted that in 3 of these there was, on the same side, a dilatation and attenuation of the walls of the middle cranial fossa. WINKELBAUER himself reports 2 cases of the same type, with demonstrated cranial changes precisely similar to the last named. One of these is of particular interest, as it is the first case in the literature, in which the cranial changes are verified by post-mortem. The case was that of a man, twenty-four years old, with enormously developed congenital, lobulate elephantiasis of the right side of the face. On section, there was found on the same side an enlargement of the middle cranial fossa, the walls of which were paper-thin, atrophic, and entirely without bone tissue in the anterior and medial walls. On the right side, too, there was an asymmetrical enlargement of the sella, and extensive atrophy of the facial bones. The hypophysis was normal; the right temporal lobe rather plump, with slightly thickened membranes, but otherwise without any tumor or congenital malformations. Finally, there was a moderate degree of internal hydrocephalus.

After a survey of the literature, WINKELBAUER comes to the conclusion that the hypophysis has nothing directly to do with the enlargement of the sella turcica in Recklinghausen's disease, and that its enlargement in these cases therefore cannot with certainty be taken as indication

of a hypophyseal disorder, but must be linked up with unilateral changes in the sphenoidal bone. He suggests two possible explanations of the cranial changes: 1) primary embryonal disturbance involving both the cerebrum and the skull and face; 2) primary local giant-growth of the cerebrum — in the reported case of the left temporal lobe — with resulting cranial anomaly owing to local increased brain pressure. He believes the latter explanation to be the more likely one, as it would be less natural to consider the cranial changes as directly due to the facial hypertrophy.

A different conception was given expression to, in 1927, in a paper by HEINE, in which he reports a yet more monstrous case of unilateral facial elephantiasis with corresponding atrophic cranial changes demonstrated by roentgen and subsequently verified by post-mortem. Figs. 12 and 13, which are borrowed from the paper referred to, give the best impression of the facial and cranial changes. The hypophysis was normal. Two cysts, corresponding to the left temporal lobe, could be explained as due either to emollition or, possibly, to abnormal traction on the cerebrum, which was otherwise normal. The meninges were slightly thickened, and firmly adherent to the membranous tissue, which in the middle and anterior cranial fossa took the place of the cranial wall. HEINE finds it most natural to see a causal connexion between the facial deformity and the atrophy of the cranial bones. He suggests three possible explanations: 1) primary lack of osseous development corresponding to the affected region; 2) deficient development, manifesting itself by disturbances of the ossification, and where the deformation of the bones is due to abnormal traction or pressure exerted on the soft, developing bone by the facial deformity; or, 3) that the osseous changes are secondary defects, which do not arise until the normal ossification is terminated. They would then be the result of an abnormal traction or pressure wielded either, through the periosteum, by the facial deformity, or, directly, either by a single tumor (neurofibroma) or several (plexiform neurofibroma), upon the still young and therefore easily impressible bone system. The first of these explanations HEINE himself sets aside as on all accounts unlikely, basing himself on BROMANN's conception of the cranium's development as dependent on that of the brain; while, in the case which he describes, the latter did not show any signs of anomalous development. The second and third are both credible; but HEINE finds it difficult to decide which of them is the most likely. The third would seem to be supported by a case reported, in 1927, by FRANGENHEIM, of a patient in whom a deformity of the right side of the face did not develop until after the eighteenth year. A plastic operation was followed by the patient's death, and the examination post mortem showed pronounced atrophic bone changes on the deformed side, corresponding to the middle cranial fossa, with dilatation of

Fig. 12.

Fig. 13.

the latter and asymmetrical enlargement of the sella turcica. Besides, there was considerable atrophic dilatation of the right orbit, in the roof and posterior wall of which there were defects, through which the cerebrum pulsated. In this case, the facial hypertrophy had begun as a lobulate hypertrophy of the contents of the orbit, after the eyeball had been extirpated following a traumatic lesion; and this furnished a natural explanation of the dilatation of the orbit.

Also a case reported, in 1927, by AVIZONIS may be mentioned as illustration of the cranial changes in Recklinghausen's disease; especially as it is exactly similar to two of those described in the foregoing (nos. 4 and 5). The patient was a boy, 9 years old. The facial and cranial changes, and the corresponding changes in the cerebrum, will be seen in Figs. 14, 15 and 16, reproduced from AVIZONIS's paper. In the right eyelid there were nodules and string-like formations, which continued as a pedicle laterally into the orbit, under the roof of the latter. By operation on the eyelid, pieces of this pedicle, as much as 10 cm. long, were removed. They were worm-like, as thick

Fig. 14.

Fig. 15.

Fig. 16.

as a lead-pencil, and extended far into the orbit. Microscopy showed a typical plexiform neurofibroma. The bulbus was normal, but displaced downwards and forwards in the orbit, and showed pulsation. As Fig. 15 shows, the middle cranial fossa is enormously dilated on the right side, with corresponding asymmetrical enlargement of the sella turcica. The bone walls were thin as paper and partly membranous, specially between the middle cranial fossa and the orbit. There was a diffuse, very considerable thickening of the meninges corresponding to the anterior pole of the right temporal lobe, with resulting increase of the dimensions of the latter. There is no record of microscopical examination of the thickened meninges; nor does AVIZONIS make any attempt to explain the changes described, as he only reports the case in order to show that the enlargement of the sella turcica could not be due to any affection of the hypophysis, as the latter was normal.

If we attempt to explain this case, it will be natural to connect the dilatation of the orbit with the existence there of the plexiform neurofibroma, which may also have been the cause of the defects in the posterior wall of the latter, through which the pulsations may have been communicated from the cerebrum to the bulbus. The cause of the dilatation of the middle cranial fossa may be found either — in accordance with HEINE — in the facial deformity; or possibly in the pressure of the cerebrum on the atrophic walls, due to neuromas in the orbit.

As a last reference to the literature, I may mention 3 cases reported in 1936 by FABEROW. They were all of the same type as those already described; and also FABEROW would see a physical causal connexion between the cranial changes and the simultaneously existing facial deformity.

I believe that with the last five cases here reported by me in this paper it is possible to illustrate the most characteristic atrophic cranial changes. All the patients had Recklinghausen's neurofibromatosis, supposedly congenital, with pigmentations of the skin; and at the same time they had, though in different degree, the disease in that special form which manifests itself in facial hypertrophy of the trigeminal region. Finally, they all presented corresponding atrophic changes of the cranium.

The cranial changes in the first three cases are all examples of secondary defects closely corresponding to the affection of the face, and — in accordance with either of the two last explanations suggested by HEINE (2 and 3) — arisen as result of the latter. In case no. 1, this must be the explanation of the atrophy of the facial bones and temporal bone of the left side; while the diastasis corresponding to the left foramen lacerum, the atrophy of the left pars petrosa, the asymmetry of the left half of the mandible and the paralysis of the left side of the glossopharyngeus muscle can be explained by the assumption of a neurofibroma on the under surface of the base of the skull. The deafness on the left ear must be explained as the result of the demonstrated changes in the middle ear following an earlier otitis, which must be supposed to have caused more serious and lasting changes in the atrophied temporal bone than is generally the case. There is nothing to support the supposition of a neurinoma of the acusticus. Finally, the considerable spina bifida of the atlas must be supposed to be a congenital malformation.

In the same way it is reasonable in the two other cases (nos. 2 and 3) to assume a causal connexion between the facial deformity on the one hand, and, on the other, the atrophic changes corresponding to the lateral margin of the orbit, the deformation of the latter, and the extensive atrophy of the bones of the temporal and parietal regions. That the latter must indeed be subject to abnormal pressure or traction seems further indicated by the fact that in one of the cases the atrophy was localised to the external side of the bones. The atrophy of the right half of the mandible and right ala magna in case no. 2, and of the left mastoid process in no. 3, clearly show the connexion between the cranial and facial changes.

Of all these three cases it may be said that both the facial atrophy and the atrophic cranial changes are moderate; and beyond the attenuation of the bones of the temporal region and, in one case, the effacement of the structure of the right ala magna, there is no enlargement

of the middle cranial fossa on the affected side, with attenuation of the walls; no asymmetry of the sella turcica, and no enlargement of the orbit, such as in the two last cases (nos. 4 and 5). The facial changes in these are dissimilar. In one there is pronounced hypertrophy of the eyelid, but only slight hypertrophy of the face; in the other there is very considerable, diffuse hypertrophy of the face, involving also the eyelid. The enlargement of the orbit in these cases can hardly be due to abnormal traction from the facial deformity, because, as we have seen, there was no such enlargement in the three others. It is therefore more reasonable to connect it with the considerable hypertrophy of the eyelid, which is a feature in both cases, and to explain it as the result of abnormal pressure from a neurofibroma in the orbit, similarly to the cases described by FRANGENHEIM and AVIZONIS. What also speaks for the correctness of this explanation is the fact that in one of the cases nodular and string-like neurofibromas were found laterally under the roof of the orbit. The pulsation of the bulbus observed in one of the cases must have been communicated from the cerebrum, through the defect in the posterior wall of the orbit; the reduced vision in the other case is explained by the demonstrated dislocation of the bulbus.

The atrophy of the walls of the middle cranial fossa may perhaps in case no. 5 be explained, in accordance with HEINE's theory, by abnormal traction on the part of the very considerable facial deformity. In case no. 4, where the facial hypertrophy is slight, this explanation will hardly suffice. Besides, in the first three cases, where there was marked facial hypertrophy, the atrophy of bones was confined to those of the temporal region and the face. It is therefore more reasonable, perhaps, to seek the explanation of the dilatation of the middle cranial fossa, and specially of the atrophy of its anterior and medial walls, with the unilateral changes of the sella turcica, in the development of a neurofibroma farther back in the orbit, with attenuation and defects in the walls and dislocation of the latter owing to the weight of the brain.

The isolated bulge in the temporal region, in case no. 4, must be considered in relation to the condition disclosed by the operation. If we consider that condition to be the result of a subsided arachnoiditis, it is possible to imagine that the latter may have caused a dilatation, which has resulted in the permanent bulge in the temporal bone.

SUMMARY

The author describes the roentgenologically diagnosed cranial changes in 8 patients with clinical symptoms of Recklinghausen's neurofibromatosis: in 2 cases dilatation of both optic foramina, with oligodendrocytoma of the optic chiasm, in 1 case abnormal vascular design in the cranial wall and depression of the sella turcica with diagnosed

arachnoiditis; in 3 cases unilateral, secondary atrophic cranial changes, corresponding to facial hypertrophy of the same side; finally, in 2 similar cases, unilateral enlargement of the orbit, the middle cranial fossa (specially the anterior and medial walls), and the sella turcica.

The opinions expressed in the literature concerning the pathogenesis are reviewed, and on the basis of these the changes in the reported cases are discussed. In regard to two of them, the author believes that the development of neurofibromas in the orbit, with thinning and dislocation of the posterior wall of the latter, due to the weight of the brain, has been a contributive factor.

ZUSAMMENFASSUNG

Verf. beschreibt die röntgenologisch diagnostizierten Schädelveränderungen bei 8 Patienten mit klinischen Symptomen der Recklinghausenschen Neurofibromatose; in 2 Fällen Dilatation der beiden Foramina optica, mit Oligodendrozytom des Chiasma opticum, in 1 Fall abnorme Gefässzeichnung in der Schädelwand und Depression der Sella turcica bei diagnostizierter Arachnoiditis; bei 3 Fällen einseitige sekundäre atrophische Kraniumveränderungen, entsprechend der Gesichtshypertrophie derselben Seite, schliesslich in 2 ähnlichen Fällen einseitige Erweiterung der Orbita, der mittleren Schädelgrube (besonders betreffs der vorderen und mittleren Wand) und der Sella turcica.

Verf. referiert die in der Literatur vertretenen Ansichten über die Pathogenese und erörtert, von diesen ausgehend, die Veränderungen in den mitgeteilten Fällen. Bezüglich zweier von ihnen meint Verf., dass Neurofibromentwicklung in der Augenhöhle mit Verdünnung und Dislokation der hinteren Orbitawand infolge des Gehirngewichtes ein beitragender Faktor war.

RÉSUMÉ

L'auteur décrit les altérations craniennes, diagnostiquées au roentgen, chez 8 sujets présentant les symptômes cliniques de la neurofibromatose de Recklinghausen: dans 2 cas, dilatation des deux foramina optiques, avec oligodendrocytome du chiasma optique; dans 1 cas, dessin vasculaire anormal dans la paroi cranienne et dépression de la selle turcique, avec diagnostic d'arachnoïdite; dans 3 cas, altération atrophiques unilatérales secondaires du crâne, correspondant à une hypertrophie faciale du même côté; finalement, dans 2 cas similaires, dilatation unilatérale de l'orbite, de la fosse cranienne moyenne (en particulier, les parois, antérieure et médiane) et de la selle turcique.

L'auteur passe en revue les opinions exprimées dans la littérature au sujet de la pathogénèse, et discute sur ces bases les altérations survenues dans les cas rapportés. En ce qui concerne deux d'entre eux, l'auteur estime que le développement de neurofibromes dans l'orbite, avec amincissement et dislocation de la paroi postérieure de cette dernière, en raison du poids du cerveau, a constitué un facteur contributif.

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ZUR FRAGE DER WIRBELSÄULENVERÄNDERUNGEN NACH TETANUS[1]

von

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Die Kenntnis von den nichttuberkulösen Infektionen der Wirbelsäule ist besonders in den letzten Jahren sehr erweitert worden; wir wissen heute, dass bei den meisten Infektionskrankheiten eine Beteiligung der Wirbelsäule vorkommen kann, selbst bei einem scheinbar so banalen Infekt wie der Grippe sind Herde beschrieben worden. (PUHL, SCHMORL).

Unter den genannten Gruppen nehmen die tetanischen Wirbelerkrankungen insofern eine Sonderstellung ein, als die beobachteten Veränderungen wahrscheinlich nicht infektiöser Natur sind, sondern einen ausschliesslich traumatischen Ursprung haben.

Es kommen beim Tetanus sowohl während des akuten Stadiums, als auch später, in der Rekonvalescenz, unter Umständen sogar erst nach Monaten, Erkrankungen der Wirbelsäule vor. Im akuten Stadium sind es Wirbelfrakturen, vom einfachen Abriss einer Randleiste bis zur Kompressionsfraktur. Vereinzelt liegen hierüber auch Sektionsbefunde vor. (ZUCKSCHWERDT).

Nach überstandenem Tetanus kann sich allmählich, und zwar vorwiegend bei Jugendlichen, ein Gibbus ausbilden, die sogenannte »posttetanische Kyphose».

Wie aus einer Arbeit von CIACCIA aus dem Jahre 1917 hervorgeht, waren bis zu dieser Zeit 16 Fälle von Gibbus bekannt, welche nach Tetanus aufgetreten waren. Grundlegende Untersuchungen über die posttetanischen Wirbelerkrankungen stammen von CHASIN und ZUCKSCHWERDT aus dem Jahre 1932. CHASIN fand unter 21 Fällen 60 % veränderte Wirbelsäulen. Zur Gruppe I gehören alle Fälle, bei denen eine leichte Kyphose im Bereich der oberen und mittleren Thoracalwirbel-

[1] Bei der Red. am 6. VIII. 1938 eingegangen.

säule besteht, bedingt durch eine geringe Abplattung der Brustwirbelkörper mit Andeutung von Keilform. Bei der Gruppe II ist eine starke Kyphose vorhanden, und die Wirbelkörper sind hochgradig abgeplattet, zeigen ebenfalls Keilform. Charakteristisch ist die Lokalisation und ein ungleichmässiges Befallensein der einzelnen Brustwirbel. Den Bandscheibenveränderungen misst CHASIN keine Bedeutung bei, er hat diese auch nur in einzelnen Fällen beobachten können, im Gegensatz zu ZUCKSCHWERDT, welcher in fast allen Fällen eine Beteiligung feststellen konnte.

Wie ZUCKSCHWERDT besonders hervorgehoben hat, ist beim Tetanus nicht die kleine Fraktur im Anfall das Wesentliche, sondern die Tatsache, dass, offenbar als systematisierte Erkrankung, die Spongiosa der oberen und mittleren Thoracalwirbelsäule allmählich unter der normalen Belastung während der Rekonvalescenz nachgibt, wobei in manchen Fällen ein bleibender Schaden entstehen kann. Dieser Prozess lässt sich durch eine Porose allein nicht ausreichend erklären und deshalb wird heute wohl mit Recht angenommen, dass durch die Traumatisierung der Wirbelsäule im tetanischen Anfall, klinisch und röntgenologisch nicht erkennbare Schäden entstehen, wie Störungen der Blutzirkulation, der Ernährung, sodass nach überstandener Infektion eine gewisse Minderwertigkeit des Knochens resultiert. Für diese Annahme sprechen sowohl die von ZUCKSCHWERDT beschriebenen Sektionsfälle, als auch die Tatsache, dass der Gibbus bei Jugendlichen und fast nur bei schwerem Verlauf des Tetanus mit langanhaltenden Krampfzuständen der Rückenmuskulatur, vorkommt. — Bezüglich der Differentialdiagnose kann in erster Linie die Abgrenzung gegenüber dem Morbus Scheuermann Schwierigkeiten bereiten. Auch hierauf hat besonders ZUCKSCHWERDT hingewiesen, weil in seinem Material Fälle vorkamen, welche sowohl klinisch als auch röntgenologisch, ohne Berücksichtigung der Anamnese, das typische Bild der »Juvenilen Kyphose» zeigten. Es ist dies auch insofern nicht zu verwundern, als SCHEUERMANN selbst Fälle beschrieben hat, welche nach einmaliger Überlastung der Wirbelsäule zustandegekommen sind. In der Regel aber unterscheiden sich die posttetanischen Kyphosen vom Morbus SCHEUERMANN, indem vorwiegend nur unregelmässig verteilt, manchmal auch nur lokalisiert, ein oder mehrere Brustwirbelkörper befallen werden.

Bezüglich der Ätiologie hat man in vereinzelten Fällen (EBERSTADT, ERLACHER, WILHELM) von spezifischer Toxinwirkung gesprochen. In Analogie mit anderen Infektionskrankheiten der Wirbelsäule, so z. B. des Typhus oder der Gonorrhoe sind bisher beim Tetanus noch niemals metastatische Herde gefunden worden, immer handelte es sich um traumatische Schäden der Wirbelkörper oder der Bandscheiben; es ist auch unwahrscheinlich, dass sich eine Toxinwirkung nur gerade an der oberen und mittleren Brustwirbelsäule geltend machen soll, während andere

Ske ettpartien, welche einer ebensolchen oder noch stärkeren Belastung aus esetzt sind, verschont bleiben.

rbeiten der letzten Jahre über Wirbelsäulenveränderungen nach Tet nus (CLARENZ, GRASHEY, NAGY, POKORNY, PORTMANN, VIGANO, WI ERSTEIN) veranlassten uns, in Gemeinschaft mit der Berner Chirurgis en Klinik, unsere erreichbaren Fälle der letzten 18 Jahre nachzukon ollieren. (Herrn J. WEYNERT wurde diese Arbeit als Dissertation übe geben). Man hat besonders darauf hingewiesen, dass nicht allein in theo etischer Beziehung, sondern auch praktisch für den Kliniker und Unf llbegutachter die Frage der posttetanischen Wirbelerkrankungen wic ig ist. Welche Unklarheit noch heute über die Häufigkeit des Vorkom nens der posttetanischen Kyphose besteht, das mag aus der Tatsach ersichtlich sein, dass die Angaben hierüber im Schrifttum zwischen 20 und 84 % schwanken.

Wir hatten Gelegenheit, 14 geheilte Tetanusfälle der Chirurgischen Klinik, klinisch und röntgenologisch nachzukontrollieren. Davon waren 12 männlichen, 2 weiblichen Geschlechts. Die Alterszusammensetzung ist aus der folgenden Tabelle ersichtlich:

2—10	Jahre	4 Fälle
11—15	»	4 »
16—20	»	1 »
21—30	»	— »
31—40	»	2 »
41—50	»	3 »

Die Inkubation zwischen der Verletzung und den ersten Zeichen von Tetanus betrug:

bis 5	Tage	2 Fälle
6—10	»	5 »
11—15	»	4 »
16—20	»	2 »
21—25	»	1 »

Auch wir haben die bekannte Beobachtung gemacht, dass der Verlauf des Tetanus um so schwerer ist, je kürzer die Inkubation war.

Bei den 14 Fällen handelte es sich 9 mal um einen sehr schweren Verlauf, mit länger als 2 Wochen anhaltenden, sich ständig wiederholenden Krampfzuständen mit Opisthotonus, 4 mal um mittelschwere Fälle, und bei einem Patienten kam nur ein Kopftetanus zum Ausbruch.

Die Therapie bestand in der Regel in Verabfolgung von Magn. sulf. intralumbal, von Serum; in einigen Fällen wurde Chloralhydrat, Somnifen, Pantopon gegeben.

Die Nachkontrollen erfolgten

nach	2	Jahren	in 3	Fällen
»	3	»	» 2	»
»	6	»	» 2	»
»	8	»	» 1	»
»	9	»	» 1	»
»	12	»	» 1	»
»	13	»	» 2	»
»	17	»	» 2	»

Die *klinische* Untersuchung ergab bei 5 Patienten einen normalen Befund im Bereich der Wirbelsäule, alle übrigen wiesen leichte Veränderungen auf, im Sinne einer geringen Skoliose oder Kyphose der Wirbelsäule. Nur in einem Fall, bei einer 43 jährigen Patientin, konnten wir eine eingeschränkte und schmerzhafte Beweglichkeit der Wirbelsäule feststellen. Bei der *röntgenologischen* Untersuchung fanden wir bis auf 2 Fälle, bei allen Patienten einen pathologischen Befund, welcher, mit Ausnahme eines Falles, unseres Erachtens nicht über das hinausging, was man auch sonst bei der normalen Wirbelsäule zu sehen gewohnt ist. Wir meinen damit, dass es nicht angeht, geringe Skoliosen und Kyphosen, leichte Bandscheibenveränderungen mit Schmorlschen Knötchen und Zeichen der deformierenden Spondylose dem Tetanus ohne weiteres zuzuschreiben. Es besteht kein Zweifel, dass man Mühe hat, eine Wirbelsäule beim Gesunden zu finden, welche allen Ansprüchen auf »normal« genügt.

Lediglich bei einem Patienten konnten wir einen Befund erheben, welcher für einen posttetanischen Schaden sprach.

Der 4 jährige Knabe macht 8 Tage nach einer Holzsplitterverletzung einen sehr schweren Tetanus mit lang anhaltenden Krampfzuständen während 2 Wochen durch. Spitalaufenthalt $1^1/_2$ Monate. Therapie: Chloralhydrat, Luminaletten, Serum.

Kontrolle nach $4^1/_2$ Jahren. Der Knabe hat sich gut entwickelt, klagt über keine Beschwerden. Funktion der Wirbelsäule o. B. Auffällig eine leichte Kyphose.

Das Röntgenbild zeigt eine deutliche Abplattung des 7. Thoracalwirbels um etwa $^1/_4$ des darüberliegenden. Die obere Bandscheibe ist etwas verschmälert.

In einem andern Fall, der vorher erwähnten 43 jährigen Patientin, haben wir den Eindruck, als ob ein bereits vorher bestandenes Leiden möglicherweise durch den Tetanus ungünstig beeinflusst worden ist:

Die 43 jährige Patientin macht 5 Tage nach einer Holzsplitterverletzung unter dem Nagel einen sehr schweren Tetanus durch. Therapie: Mag. sulf. intralumbal. Spitalbehandlung $1^1/_2$ Monate.

Kontrolle nach 2 Jahren. Pat. klagt über starke Rückenschmerzen, welche seit der Erkrankung aufgetreten sind; früher hat sie angeblich nie Beschwerden gehabt. Die Beweglichkeit der Wirbelsäule ist eingeschränkt, besonders im Bereich der unteren Thoracalwirbelsäule.

Abb. 1. Wirbelsäule eines $8^1/_2$ jährigen Knaben, $4^1/_2$ Jahre nach Tetanus. Isolierte Abplattung des 7. Thoracalwirbels, obere Bandscheibe verschmälert.

Abb. 2. Derselbe Fall, Profilbild.

Das Röntgenbild zeigt eine sehr ausgesprochene S förmige Skoliose der Wirbelsäule und eine Spondylose, welche über das Mass hinausgeht, was man sonst im Alter von 43 Jahren findet. Ferner bestehen Schmorlsche Knötchen und eine »bajonettförmige« Abkrümmung in der Gegend des 10. Thoracalwirbels.

Erwähnt sei noch kurz der schwerste Fall, welcher mit Erfolg in der Klinik behandelt worden ist. Hier trat nach 3tägiger Inkubation, bei

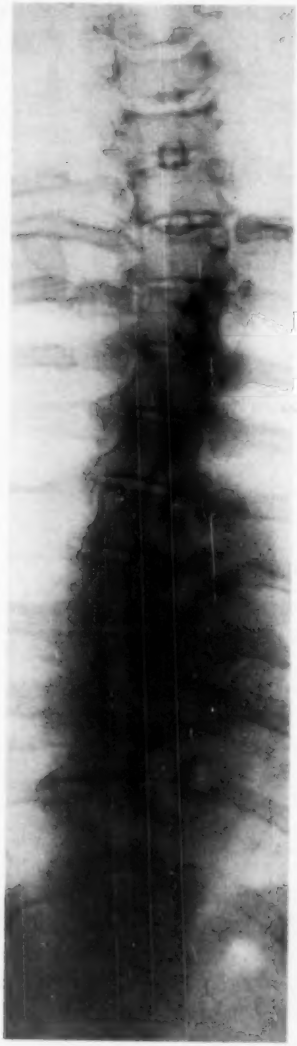

Abb. 3. Wirbelsäule einer 45 jährigen Patientin, 2 Jahre nach Tetanus. S förmige Skoliose der Wirbelsäule, z. T. hochgradige deformierende Spondylose, Bandscheibenschäden, »bajonettförmige« Abknickung der Wirbelsäule bei Th. 10. Fragliche Verschlimmerung einer schon früher bestandenen Erkrankung der Wirbelsäule durch Tetanus.

Abb. 4. Derselbe Fall, Profilbild

eine 19 Jährigen mit einer Schnittverletzung am Kopf ein ausserordentlich heftiger Tetanus auf, bei dem erst nach 23 Tagen die Krampfanfälle aufhörten. Die Kontrolle, welche nach 13 Jahren vorgenommen wurde, ergab einen ganz normalen Befund im Bereich der Wirbelsäule; der Pat., war auch völlig beschwerdefrei.

Wir kommen also zu dem etwas überraschenden Ergebnis, nur in 7 % von 14 Fällen einen posttetanischen Schaden gefunden zu haben.

Abgesehen von der verschiedenen Zusammensetzung des betreffenden Materials, der geringen Anzahl der bisher nachuntersuchten Fälle, haben wir den Eindruck, dass gelegentlich die Häufigkeit des Vorkommens der posttetanischen Kyphose überschätzt wurde, indem man Befunde dem Tetanus zuschrieb, bei denen der Beweis für die spezifische Ätiologie nicht erbracht war. Auf der andern Seite sollte sowohl der Kliniker, als auch der pathologische Anatom, mehr wie bisher, auf die Wirbelsäule achten; ebenso sind die Befunde für den Unfallbegutachter wichtig, wenn es sich um die Beurteilung von Spätschäden nach Tetanus handelt.

ZUSAMMENFASSUNG

Es wurden 14 Fälle von überstandenem Tetanus klinisch und röntgenologisch nachkontrolliert. Nur in einem Fall, bei einem 8 jährigen Knaben, der $2^1/_2$ Jahre vorher einen sehr schweren Tetanus durchgemacht hatte, konnte ein Befund an der Wirbelsäule erhoben werden, der für einen posttetanischen Schaden charakteristisch war. Es bestand eine isolierte Abplattung des 7. Thoracalwirbels mit geringer Beteiligung der angrenzenden Bandscheiben. Bei einem anderen Fall musste die Verschlimmerung einer schon vorher bestandenen Spondylose angenommen werden. Alle übrigen Fälle wiesen Veränderungen auf, die nicht über das Normalmass hinausgingen, sodass die Häufigkeit der posttetanischen Wirbelsäulenveränderungen nur 7 % betrug.

SUMMARY

Fourteen patients who had recovered from tetanus were subjected to clinical and roentgenological control. In only one, a boy of 8 years who had survived severe tetanus $2^1/_2$ years earlier, was it possible to demonstrate any change in the vertebral column which could be regarded as characteristically post-tetanic. It consisted of an isolated flattening of the 7th thoracic vertebra with slight participation of the adjacent intervertebral discs. Advancement of a spondylo-arthrosis which had been present before the tetanus had to be assumed in another instance. All the other cases showed lesions not beyond the normal so that the frequency of the post-tetanic alterations amounted to only 7 %.

RÉSUMÉ

14 sujets, rétablis du tétanos, furent soumis à un contrôle clinique et roentgenologique. Dans un seul cas, celui d'un garçonnet de 8 ans ayant subi 2 ans $^1/_2$ auparavant un tétanos particulièrement grave, il fut possible de démontrer une altération de la colonne vertébrale pouvant être considérée comme lésion post-tétanique caractéristique. Elle con-

sistait en un aplatissement isolé de la 7e vertèbre thoracique, avec légère participation des disques intervertébraux adjacents. Dans un autre cas, il y eut lieu de présumer une aggravation d'une spondylo-arthrose préexistante. Tous les autres cas présentèrent des lésions ne dépassant pas la normale, de sorte que la fréquence des altérations vertébrales post-tétaniques ne s'est élevée qu'à 7 %.

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ON THE DIVISION OF THE LUNG SEGMENTS IN THE RIGHT UPPER LOBE[1]

by

Dr. *E. Behr* and Dr. *Eelco Huizinga.*

Owing to various circumstances, to the development of pulmonary surgery and of bronchoscopics, to the improvement in the technique of radiology and of bronchographics, the need has made itself felt, and it has become possible, to locate more exactly certain affections in the lungs of patients. An attempt has been made to find a connection between the seat of the affections and the anatomical construction of the lungs. Names such as »upper, middle, and lower field», wich are generally used in röntgenology, are suitable for indicating an affection which is visible on a röntgen photograph, but do not take the anatomical factors sufficiently into consideration. An affection in the right upper field, for instance, may in different circumstances be located both in the upper, middle, and lower lobe.

It is better, in every possible respect, to keep to a division according to the lobes of the lung: three on the right, two on the left. Each of these is always served by its own bronchus. It is obvious that, with a further division of the various lobes, the ramifications of the bronchus in question are determined. There are always a few large branches, each branch serves a particular part of a lung lobe, which is frequently termed a »lung segment». A few points are of great importance in this connection:

1. The manner in which the bronchial tree and the pulmonary artery ramify is, according to many investigators, the same, a particular part of the lung is therefore served by a corresponding bronchus and artery. In this way a lung segment becomes to a still greater extent a unit, not only in physiological, but also in pathological respect. It is known that many pulmonary affections, such as atelectasis, pneumonia, abscess,

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infarct, bronchiectasis and also tuberculosis, may, and indeed frequently must, confine themselves to one or several lung segments. It would thus be possible to account for this by either a bronchogenous or a hematogenous origin, with which the cause of the affection had proceeded from one particular supply branch of a bronchus or pulmonary artery.

2. Both the bronchi and the arteries are terminal ramifications, a sharply defined part of the lung is therefore served only by that particular branch.

3. The lung segment is always wedge-shaped, the point is central, where the large branches ramify, the base is directed towards the surface. The base of almost all lung segments reaches the periphery of the lung in such a way that this, as it were, is projected on to the wall of the thorax. This is of practical significance, for instance for the surgical treatment of the pulmonary abscess.

4. Not until a clear idea had been formed of the segmental construction of the lung was it possible to account in the right way for very remarkable shadows found in röntgenphotographs of many patients. A good example of this is the »directed Atelectasis» of the lung (FLEISCHNER (1)). In röntgeninvestigations horizontal strips are repeatedly found in the *lowest* pulmonary field, usually in the case of abdominal patients who were serously ill. It was considered possible that these were due to a conducted pleuritis or pneumonia. In reality, however, the affection is to be accounted for by the atelectatic collapse of a particular part of the lung owing to stoppage of the bronchus by secretion, with impaired respiration. As a result of stoppage of a large bronchial ramification a determinated lung segment collapses, whilst the wall of the thorax and the hilus remain in the same position; the segment can therefore only become smaller in the upward and downward direction, and so becomes a flat disc. Strip-like shadows of this kind, running from the pulmonary hilus to the periphery, have also been described in the right upper lobe. It was then found that the branch in question is usually obstructed by tuberculous hilus glands. Excellent illustrations are to be found in WESTERMARK's publication (2):

The importance of the following question is obvious: is the anatomy of the lung of such a nature that a fixed system is to be found in the division of the pulmonary segments? If so, it would then, in a particular case with a circumscribed affection on the thorax photographs, which of course should be taken in various directions, be possible to state immediately and with certainty in which lung segment the aberration is located. As it is known that each lung segment has its own bronchus service, this really comes back to the old problem which so occupied AEBY (3): can a definite scheme of the bronchial ramifications be given?

Various schemes for the lung segments have been drawn up, which may best be distinguished according to the different languages in which this has been done. There is, for instance, a German scheme according to FELIX (4), in which a slight modification has recently been introduced by HERRNHEISER (5), a French one according to LUCIEN (6), and an Anglo-Saxon one by KRAMER and GLASS (7). It is remarkable that these schemes agree much less well than would a priori be expected. Of course, there is in all cases the same division into lung lobes, but the division into segments varies very much. In the German scheme, for instance, three segments are given in the right upper lobe, in the French and American schemes four.

Which, then, of these schemes is the correct one? In point of fact not one of them is of general validity, since the division of the lung segments is not in all cases the same. This changes, in fact, with the anatomical variations of the bronchial tree, which was not taken sufficiently into account by the various investigators. Here, too, however, as is always the case in anatomy, there is one type which occurs most frequently. In order to form an independent opinion as to this, we carried out an investigation of the lungs obtained from 108 dissections. In this communication we shall confine ourselves to the results with regard to the *right upper lobe.*

A very good impression of the division of the segments may be obtained by injecting paraffin with a low melting-point into the ramifications of the bronchus; we used a quality of from 40 to 45° C. for this. The paraffin, which had been liquefied by heating, was injected by means of an ear-syringe, after a well-fitting ear-funnel had been introduced into the branch to be filled, whilst the rest were temporarily closed by means of wads of cottonwool. The paraffin was mixed with a different colouring matter for each of the various ramifications, Prussian blue and »imitation carmine» being very satisfactory. The use of colouring matters which easily dissolve, such as eosine, is not to be recommended, as the preparation is thereby more or less entirely coloured, the otherwise so conspicuous sharp boundaries of the segments then becoming vaguer. After fixation the injected lung lobe is later divided into slices.

Fig. 1 is a schematic, accurate drawing of one of these slices, which was obtained by making frontal incisions through the upper lobe of the right lung of a girl of 17 years of age. We see here 3 segments with sharp boundaries, all typically wedge-shaped, with the point directed towards the centre and the bases towards the periphery. An examination of this illustration enables one to understand how, when a segment collapses, the strip-shaped shadows to be seen on WESTERMARK's röntgen photos must be formed. The uppermost segment, the apical one, which is hatched horizontally, comprises the entire tip of the lung; it runs with

a point downwards. Sometimes it reaches to the interlobary fissure between the upper and the middle lobe. In this case, however, this segment is comparatively small, whilst the vertically hatched lowest medial, the pectoral one, is the largest. It comprises the lowest, front and medial part of the upper lobe. The rest, the dorsal and lateral part, is taken up by the smallest axillary segment, which is hatched diagonally.

The bronchus of the upper lobe of the right lung therefore divides here into 3 ramifications. According to HERRNHEISER, who made a special study of these relations, this is the most frequent type. DWIGHT DAVIS (8), on the other hand, considers that a division into two is most commonly found.

Our experience agrees with that of HERRNHEISER, for our investigation showed beyond a doubt that the division into three is strongly predominant. In 108 cases this was found 63 times, whilst in 45 cases there was a division into two. In this latter group, however, there were 7 cases in which one branch so soon ramified again as to give rather the impression of a somewhat bungled division into three. The other 38 cases of division into two may be divided into three groups:

1. A bifurcation was found in 26 cases, the partition being more or less in the frontal surface. There is, therefore, an anterior and a posterior branch. This latter varies greatly in size, but is invariably smaller than the anterior one. In a number of the cases this posterior branch is exactly like the axillary branch of the trifurcation. Here, too, this branch soon bifurcates, one branch behind, which serves the dorsal paravertebral part of the lung, and a medial branch running laterally to serve the axillary part. The foremost and larger branch comprises the apical and the pectoral of the trifurcation, but there are frequently considerable differences in the way in which the further ramification occurs.

In another part the posterior branch is much wider, and from this branches proceed upwards as far as the tip of the lung, so that the upper lobe can then be divided by a vertical plane into an anterior and a posterior half.

2. In 9 cases there was a bifurcation with a horizontal partition. There is then a lower and an upper branch, both of which as a rule soon bifurcate through a vertical partition. In this way an entirely different anatomical relation of the upper lobe is produced. This is divided by a horizontal plane into an upper and lower segment, or, if we start from the bifurcation of the main branches, which soon follows, into two upper and two lower segments.

3. In 3 cases a small branch soon branched off upwards, hardly any further than the main bronchus which serves the medial portion of the pulmonary apex. The bronchus of the upper lobe divides further up into

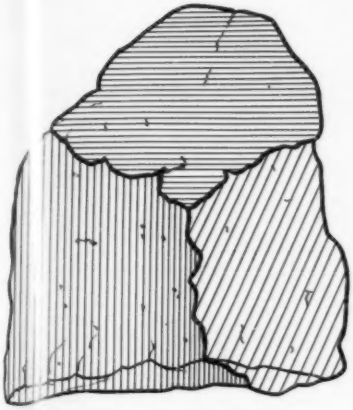


Fig. 1. Frontal cut through the upper lobe of the right lung; girl of 17 years. In the original preparation the branches of the bronchus of the upper lobe were injected with differently coloured paraffin. The boundaries of the segments are very sharply defined.

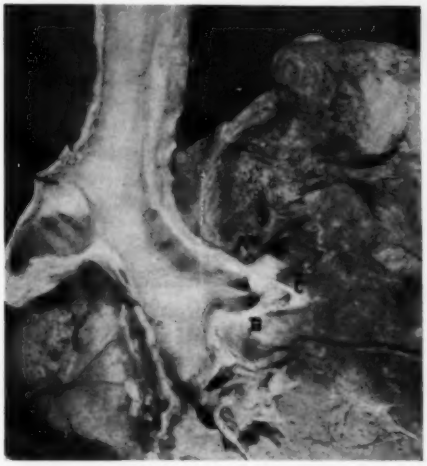
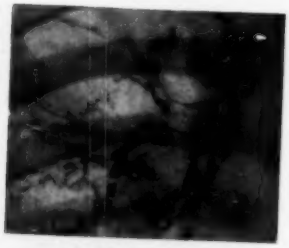


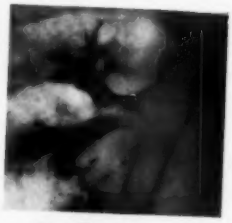
Fig. 2. Lung preparation, in which trachea, main bronchus and bronchus of the right upper lobe have been cut open from behind. Man of 59 years of age.
Typical trifurcation right upper lobe bronchus.
A. = apical branch upward.
B. = pectoral branch forward.
C. = axillary branch lateral.

Fig. 3. As fig. 2. Woman of 41, Bifurcation right upper lobe bronchus in anterior and posterior branch through a frontal partition.

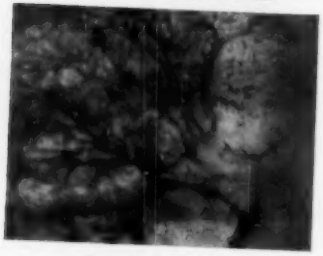
Fig. 4. As fig. 2. Man of 61. Bifurcation right upper lobe bronchus with horizontal partition. Upper and lower branch soon bifurcate, both vertical partitions to be seen in the depth.



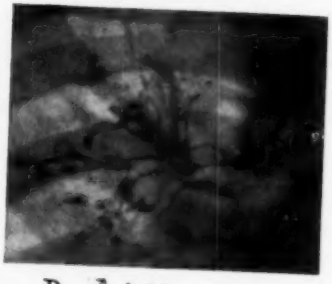
A ♂ 2 Years



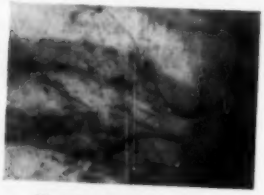
B ♂ 2 Years



C ♂ 9 Years



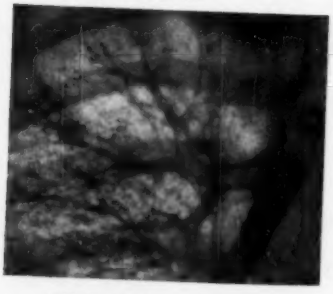
D ♂ 4 Years



E ♀ 2 Years

F ♀ 6 Years

G ♂ 27 Years



H ♀ 3 Years

Fig. 5 A—H.

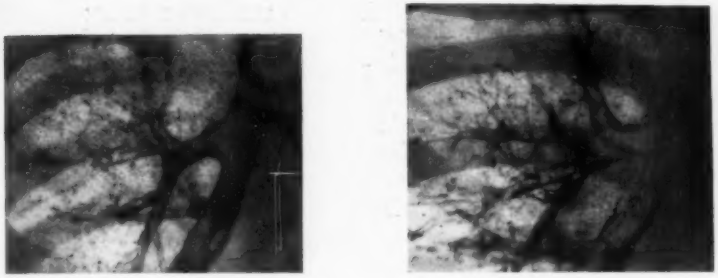


Fig. 5 I—J.

Fig. 5. Röntgen photographs of the bronchus ramifications of the right upper lobe after lipiodol filling. For explanation, see text.

two, a pectoral and an axillary branch, which also serve the lateral portion of the pulmonary apex.

Figures 2, 3, and 4 show the most important types. They are photographs of dissection preparations, the trachea and the right upper lobe bronchus being cut open behind far enough plainly to show the ramification of this latter. Fig. 2 shows the usual trifurcation; the preparation is from a man of 59 years of age. The apical branch can be followed upwards for a moment; under this is seen the pectoral branch, whilst the axillary goes off laterally. Fig. 3 is a bifurcation of a vertical partition; the posterior branch in this case is wide, and serves part of the pulmonary apex also. It is the right upper lobe of a woman of 41. Fig. 4 shows quite a different picture again; the bronchus of the upper lobe of the right lung of a man of 61 is here divided by a horizontal partition into two parts; it is clearly to be seen how both branches soon split dichotomically through a vertical partition.

From this investigation it is seen that the ramifications of the bronchial tree are rich in anatomical variations. As was to be expected, and was also found in our cases, this holds good for all ages, from newborn babies to very old people. Moreover, in ramifications of the same type, considerable differences exist in the width and consequently also in the extent of particular branches. With the ordinary trifurcation the axillary branch is usually the smallest, but one of the other two branches may also be so; the width-proportions are subject to great variation. A particular part of the lung may thus in different cases be served by quite a different branch. This is also to be seen in lipiodol photographs, of which many hundreds have been made at Groningen.

Fig. 5 shows the röntgen photographs of 10 bronchi of the right upper lobe, almost all of children, since here the bronchial tree is as a

rule better filled with contrast oil into the smallest ramifications than is the case with adults. They are lipiodol photographs selected from a large collection to show the differences plainly.

A and B are examples of a bifurcation into equal branches, the course of which is plainly to be followed for a certain distance, until a furcation into several smaller branches takes place. Type C may be regarded as a dichotomy, the two branches soon splitting again into two, so that 4 large branches are to be seen. In D there are as many as 5 branches; presumably, however, this is a trifurcation, in which the uppermost apical branch soon parts into three.

E—J are photographs of the ordinary type, the trifurcation. Here, too, a great variation in the course and the calibre of the various branches is to be seen. In the case of E there is a uniform division into 3 equal branches: the uppermost one, the apical, serves the entire apex of the lung; the middle one runs off to the side, the axillary; the lowest one, the pectoral, as is seen in transverse photographs, also goes strongly forwards. In F—J these 3 branches are in all cases easy to find, but the topography is in every case different. In F the apical branch is the smallest; usually this is the largest, as in G. In H the apical branch is still smaller; here it is noticeable that the largest lateral portion of the pulmonary apex is now served by the abnormally large axillary branch. As a rule the axillary branch is the smallest, as in I. Here, as also in J, the pectoral branch predominates.

It follows from the above that it is not possible to indicate a scheme for the division of the lungsegments, as various investigators have tried to do. This does not alter the fact that the study of the manner in which the lung is divided has done very much to give a better insight into particular cases of disease, and that a knowledge of the chief anatomical variations in the ramification of the bronchial tree is of importance to everyone who is interested in the pathology of the respiratory tract and of the lungs. With a particular case of disease it is not possible, if a knowledge of the anatomical conditions be required, to rely upon a theoretical scheme but it will be necessary to carry out a bronchography.

SUMMARY

A bronchial branch serves a sharply defined portion of lung: a lung segment. The extent of this can be shown very well by injecting dissection preparations with paraffin. The various pulmonary lobes can be divided into a particular number of segments. The importance of this in hospital work is pointed out. Various investigators have endeavoured to indicate a fixed scheme for this division, which depends on the manner in which the bronchial tree ramifies. An investigation of the right upper lobe of 108 lungs showed this to be impossibel. A particular type of ramification, a trifurcation, does, however, occur most frequently, but the anatomical variations are numerous. This again points to the utility of bronchography in a particular case of disease.

ZUSAMMENFASSUNG

Ein bestimmter Bronchialast versorgt einen scharf umschriebenen Lungenteil: ein Lungensegment. Die Ausbreitung zeigt sich sehr deutlich wenn Leichenlungen injiziert werden mit Paraffin. Die verschiedenen Lungenlappen können verteilt werden in einer bestimmten Anzahl von Segmenten. Die klinische Bedeutung davon wird besprochen. Einige Untersucher haben versucht ein festes System für diese Verteilung anzugeben. Das hängt also zusammen mit der Frage der Verästelung des Bronchialbaumes. Durch die Untersuchung des rechten Oberlappens bei 108 Lungen wurde festgestellt, dass kein festes Schema besteht. Zwar überwiegt eine bestimmte Art von Verästelung stark, bei diesem Oberlappen ist das eine Dreiteilung, aber es bestehen viele anatomische Variationen. Das beweist wieder den Nutzen der Bronchographie bei einem bestimmten Fall in der Klinik, wobei eine genauere Lokalisation gewünscht wird.

RÉSUMÉ

Une ramification bronchique alimente une partie très nettement limitée du poumon: un segment pulmonaire. L'extension de ce segment est très bien démontrée par l'injection de paraffine dans des préparations anatomiques. Les différents lobes pulmonaires peuvent être partagés en un nombre déterminé de segments. Les auteurs font remarquer l'importance de ce fait pour la clinique. On a essayé de divers côtés de donner un schéma fixe de cette division qui dépend de la façon dont se produit cette ramification de l'arbre bronchique. L'examen du lobe droit supérieur de 108 poumons a montré que cela est impossible. On voit bien le plus souvent un type déterminé de ramification, mais les variations anatomiques sont très nombreuses. Ceci montre une fois de plus l'utilité de la bronchographie dans un cas déterminé de maladie.

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